# VISUO-MOTOR CONTROL FOLLOWING STROKE:

# A MOTOR SKILLS PERSPECTIVE

SINCLAIR LOUGH

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## DEDICATION

This thesis is dedicated to the memory of my father.

## DECLARATION

I declare that this thesis has been wholly composed by myself.

THE UNIVERSITY *of* EDINBURGH

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ABSTRACT

Although the residual motor dysfunction which results from stroke poses a considerable burden upon both the patient and treatment resources, little objective data has been gathered on how movement is affected. The experiments contained in this thesis offer a preliminary account on the status of visuo-motor control following stroke using techniques and theoretical constructs derived from the study of normal motor control.

The experimental work focuses on arm movement, and in particular on movement about the elbow. The results of the first series of experiments indicate the importance of visual information to a realistic appreciation of the position of the affected arm in space. In the second Chapter a kinematic analysis demonstrates that vision can act to improve accuracy, speed and smoothness of movement. On the basis of EMG data it is argued that vision operates to improve the timing of agonist/antagonist contractions. The final Chapter of experiments explores this issue further within the context of bilateral movements.

A model of dysfunction is proposed and conclusions for treatment by physiotherapy are considered.

# Introduction

The annual incidence of stroke in the U.K. is estimated at two in every thousand (Langton-Hewer, 1976) with about two thirds of the victims surviving. However about two thirds of those survivors suffer a severe residual disabilty of movement (Licht, 1975). The task of treating this disability falls largely to the physiotherapy profession and Bobath (1978) estimates that ten percent of the working day in physiotherapy departments is spent on the rehabilitation of stroke patients.

Whilst acknowledging that the motor deficit caused by stroke can be influenced by the presence of accompanying cognitive and/or linguistic deficits, this thesis concentrates on trying to elucidate the fundamental problems in motor control of the upper limb which face patient and therapist.

In Chapter I the relevant literature is reviewed. However this review is not intended to be exhaustive, as some of the topics demand a practical knowledge of techniques and procedures which the author does not possess. Also, because the experimental work in this thesis is solely concerned with the control of arm movements, the literature on hemiparetic gait is not discussed.

The review commences with a discussion on stroke and its consequences for movement. The concept of stroke as an upper motoneuron lesion is examined. Following this it is noted that little attempt has been made to scientifically study hemiparesis. Those studies which do, tend to examine abnormalities in localised reflex behaviour, rather than the control of voluntary movement. Treatment by physiotherapy is then described and found to be dominated by subjective clinical experience, instead of objectively gathered data. This then leads on to the aim of the thesis and the proposition that the effect of stroke on movement could be experimentally studied by employing the theoretical constructs and techniques used in the study of normal motor skills. The Chapter concludes with a brief review of this area of research in anticipation of issues and hypotheses raised by the experiments which follow.

Chapters II, III and IV attempt to fulfill the aim of the thesis through a comparative, behavioural analysis of affected arm movement under varying conditions. The experiments designed to do this are guided by three principal questions:-

1) What is the role of vision in the control of the hemiparetic arm?

2) What sort of communication exists between the affected and unaffected limbs?

3) Does spasticity interfere with the performance of

movement?

In these Chapters, discussion is largely restricted to hypotheses about control processes. In Chapter V, the three Chapters are summarized, tied together, and the implications for physiotherapy considered.

# Chapter I

1.1 - Stroke, hemiparesis and the nature of the lesion.

Stroke is typified by a relatively rapid onset of focal, neurological deficit persisting for longer than 24 hours and resulting in a loss of willed movement on one side of the body (hemiplegia). Whisnant (1976) outlines four diagnostic categories of stroke:-

1) Cerebral thrombosis leading to cerebral infarction.
2) Cerebral embolus leading to cerebral infarction.
3) Intracerebral haemorrhage.
4) Subarachnoid haemorrhage.

From autopsy studies (Fisher and Curry, 1965; Jörgensen and Torvik, 1969) the most common site of cerebral vascular accident (C.V.A.) can be identified as the middle cerebral artery resulting in lesion of the internal capsule and neighbouring cortical tissue.

The internal capsule is the principal route by which cortical projections are funnelled to the spinal tract via the corticospinal or pyramidal tract (Brodal, 1962). This anatomical feature, coupled with the tendency until the late 1940's to treat stroke in the same manner as poliomyelitis, led to a traditional view of hemiparetic dysfunction resulting from a pyramidal or upper motoneuron lesion and causing a loss of drive to the alpha motoneurons supplying the affected musculature.

In 1951 Twitchell embarked on a study to delineate

some of the factors involved in the recovery of movement following hemiplegia. He found:-

"..a remarkable uniformity in different cases" page 444
and
"..a general pattern in which certain phenomena predominated during distinct phases or stages of the recovery process" Twitchell (1951) p 444.
Following a short initial period of flaccidity, the first sign of a return of activity was in the form of hyperactive tendon jerks at the limb extremities. This was closely followed by an increase in resistance to passive movement in the plantar flexors of the ankle and in the palmar flexors of the wrist. The intensity of this resistance gradually increased and additionally involved the adductors and flexors in the upper limb and the adductors and extensors in the lower limb. Soon after this, the clasp-knife phenomenon appeared in the extensors of the knee and the flexors of the elbow.

The first sign of a return of movement came in the hip and the elbow flexors. Thereafter, a gradual return of flexor activity about all joints in the upper limb reappeared. However there was an inability to flex the elbow, wrist or fingers in isolation and any attempt at this resulted in a pattern of total upper limb flexion. Twitchell states that at this point in recovery, spasticity was at its most intense and could be influenced by the tonic neck and righting reflexes.

Shortly after the development of the flexor synergy

the extensor synergy of the arm appeared in a similar proximal to distal manner. As voluntary movement increased spasticity lessened and eventually finger movement became more dextrous.

Twitchell's study was largely concerned with the upper limb, although his observations on the lower limb indicated that a similar series of changes occurred, but with a dominance of extensor and not flexor activity. Twitchell divides recovery into three distinct stages:-

"..the first being dominated by proprioceptive reactions, the second by contactual stimulation of the extremity and finally a seeming total independence of movement upon such external agents." p477

It is important to note that not all of the patients studied by Twitchell made a complete recovery. In many recovery appeared to cease in the early stages. Twitchell's observations were subsequently confirmed by the clinical experiences of Brünnstrom (1970) and Bobath (1978) with some of them objectively validated by De Souza et al (1980).

As previously mentioned, capsular hemiplegia is traditionally held to result from a lesion of the pyramidal tract. However Bucy et al (1964) point out that a pure corticospinal lesion is highly unlikely in the internal capsule. It is only in the medullary pyramids (Tower, 1940) or the central portion of the cerebral peduncle (Barnard and Woolsey, 1956) that these

fibres are sufficiently segregated from all ascending fibres and other descending fibres for a lesion of this integrity to occur.

Bucy et al (1964) report the case of an operation carried out in man where the central portion of the right cerebral peduncle was divided to relieve a left hemiballismus. Immediately after the operation the patient had a complete, flaccid left hemiplegia. However within 24 hours signs of a return of voluntary movement were apparent and in marked contrast to the stroke victim, part of that early return of function occurred in the fingers and the toes. Also, as recovery progressed it was in a distal to proximal manner with little hypertonus and only moderately hyperactive tendon reflexes. Bucy et al (1964) report that the patient proceeded to make almost total recovery with little residual dysfunction in the execution of fine movements.

Two and a half years after the operation the patient died of an unrelated disease and Bucy et al (1964) had the opportunity to perform a post-mortem to examine the location and extent of the peduncular lesion and its subsequent degeneration. This confirmed that only the central portion of the right cerebral peduncle was divided and revealed that 83% of the corticospinal tract supplying the left side of the body had degenerated.

The fact that almost all the corticospinal tract had been destroyed and yet a full and rapid recovery ensued, in a distal to proximal direction, without spasticity, points to the fallacy of considering the effect of stroke as an upper motoneuron lesion. Although there may be damage to the corticospinal tract after a capsular lesion, much of the resultant motor dysfunction must be due to lesion of other cortico-fugal pathways. Support for this conclusion comes from the results of experimental surgery in primates as outlined by Lawrence and Kuypers (1968a and b).

Kuypers (1963) advanced the hypothesis that the descending pathways, composing the classical pyramidal and extrapyramidal systems, should be recombined into two more functionally, meaningful groupings on the basis of their distribution of termination in the spinal cord. The motoneurons occupy the ventral horn of the spinal cord, with those innervating the distal musculature located laterally and those innervating the axial musculature, ventromedially. The interneurons projecting to the motoneurons are located in the intermediate areas of the spinal grey and the dorsomedial region of the ventral horn. Interneurons leading to motoneurons supplying the axial musculature are in the ventromedial zone of the internuncial region with those projecting to motoneurons innervating the distal muscles located laterally. This led to the descending pathways being

grouped according to a ventromedial or lateral termination.

The ventromedial system comprises the vestibulospinal and reticulospinal tracts, whilst the lateral system is composed of rubrospinal fibres. The corticospinal tract overlaps the two systems in that it forms direct connection with motoneurons in the lateral region, and its projection at the internuncial level is both lateral and ventromedial.

In order to uncover the function of the subcortical components of the two systems Lawrence and Kuypers (1968a) performed a bilateral section of the pyramidal tracts in rhesus monkeys. They then observed the free movements of the animals during recovery. It was found that a wide range of movement rapidly recovered, but that there was a loss of individual finger movement. This contrasts with Bucy et al's observation of an early return of finger function in their patient. Commenting on this, Lawrence and Kuypers attribute it to be due to the sparing of some pyramidal fibres in the patient. The only other persistent deficit in the monkeys was a slowness of movement.

Those monkeys who recovered from pyramidal interruption were subsequently lesioned in either the ventromedial or lateral systems at various levels of the

neuroaxis. Lesion to the ventromedial system at the medullary reticular level hardly affected the distal musculature, but severely impaired axial and proximal activity and produced an alteration in the postural attitude of the trunk and limbs in the direction of flexion. There was also a loss of righting reflexes and disturbances of balance. Three of the animals in this group had been sectioned without prior pyramidectomy. They demonstrated similar, but less severe effects.

Lesion of the lateral system by destruction of the rubrospinal tract at the medullary level did not result in axial or proximal limb flexion postures, or dysfunction in balance or righting, but did produce weakness in the elbow, wrist, and hand. Lesion of the red nucleus resulted in ataxia in the neck, trunk and proximal, ipsilateral arm. In addition the posture of the arm was similar to that of the animals with lateral medullary lesions. There was also a release of tonic neck reflexes.

Lawrence and Kuypers' studies conclude that the corticospinal tract is responsible for speed and finesse of movement, particularly of the distal musculature. The lateral brainstem pathways also seem responsible for independence of distal activity and for limb flexion, whilst the ventromedial brainstem system controls posture and the integration of body and limb movement. Referring

back to the description of stroke given by Twitchell (1951), elements of dysfunction in all three of the systems outlined by Lawrence and Kuypers compose the disorder known as hemiparesis. Therefore the capsular lesion interrupts not only corticospinal projection, but also cortico-fugal interaction with the descending brainstem pathways.

## 1.2 - Experimental study of motor dysfunction following stroke

Little attempt has been made to experimentally elucidate the motor dysfunction caused by stroke. Instead work on the disorder has concentrated on the patient's ability to perform activities of daily living (ADL) rather than detailed analysis of movement (eg Lehmann et al, 1975; Andrews et al, 1982)

Most experimental studies of motor dysfunction following stroke are anchored in the observed clinical phenomena of flaccidity, enhanced tendon reflexes, and increased muscle resistance to passive stretch. Many studies focus on the phenomenon of spasticity. A generally accepted definition of spasticity has been given by Lance (1980, page 485): "Spasticity is a motor disorder characterised by a velocity-dependent increase in tonic stretch reflexes ('muscle tone'), with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neurone syndrome." Typically researchers examine activity about one joint only, often the ankle. The purpose behind this class of experimentation is twofold:-

1) An elucidation of the spinal mechanisms underlying the production of movement.

2) To further understanding about hypotonia and hypertonia.

Very often (1) is the primary goal, with (2) being a means of achieving it.

The techniques employed in this area of research may be summarised as involving stimulation of the muscle or its efferent or afferent supply and recording the motor response. This is done both invasively and non-invasively and includes the following responses:-

1) The stretch reflex - (Matthews, 1964; Burke et al, 1978; Herman et al, 1973). By stretching the muscle either statically, or by movement around the joint, the muscle spindles are stimulated and impulses from the muscle afferents result in the firing of the alpha motoneurons supplying the muscle.

2) The H reflex - (Hoffman, 1918; Tanaka, 1974; Yanagisawa et al, 1976) Through electrical stimulation of the afferent supply of the muscle at a level submaximal for efferent stimulation, the resultant impulses in the Ia fibres causes the alpha motoneurons supplying the muscle to fire.

3) The M response - (Ashby and Verrier, 1976; Yanagisawa et al, 1976). This is the compound action potential of the muscle resulting from a supramaximal electrical stimulation of the common nerve.

4) The tendon reflex - (Ashby and Verrier, 1976). Monosynaptic activation of the alpha motoneurons through a mechanical tap on the tendon.

5) The tonic vibration reflex - (Lance et al, 1973;

Hagbarth 1973; Somerville and Ashby; 1978). This is elicited by vibration of the muscle or its tendon.

In many studies these responses are not examined in isolation, but combined to try and tease out the levels of excitation and inhibition in the segmental apparatus. For example, Somerville and Ashby (1978) assume the M response to represent the electrical activity of 100% of a muscle's motor units. By comparing the compound action potential of the H reflex with this value, the proportion of the muscle motoneuron pool capable of being reflexly activated can be assessed. The H/M ratio can then be taken as a measure of central excitability. Similarly the activity induced by the tendon reflex can be compared with the M response to give a measure of both central excitability plus spindle excitability. Thus spindle excitability can be assessed by comparing the two ratios.

Magherini et al (1972) found that in animal preparations that vibration produces monosynaptic facilitation of motoneurons and inhibition of pre-synaptic inhibition of the primary afferents. Delwaide (1973) demonstrated a similar polysynaptic inhibitory effect of vibration in man. Therefore by studying the effect of vibration upon the H reflex and comparing the result with a control H reflex and the ratios outlined above, the balance of excitation and inhibition at the spinal level can be assessed.

Two alternative hypotheses run through many of the neurophysiological studies of hemiplegia and hemiparesis:-

1) That muscle spindle excitability is increased.

2) That the excitability of the alpha motoneuron is increased.

The first hypothesis was posited to account for the clinical observation that in the early stages of recovery, when the muscles are flaccid, tendon jerks are exaggerated. Buller (1957) demonstrated that in hypotonia the monosynaptic reflex was greater than normal thus implying that fusimotor drive to the muscle spindle had increased. By selective blocking of fusimotor axons with procaine, Rushworth (1960) found that in the spastic state hypertonia was decreased. Also, dorsal rhizotomy has been shown to reduce spasticity (Freeman and Heimburger, 1948). These studies and similar work by Dietrichson (1973) tend to support the first hypothesis.

Delwaide (1973) and Hagbarth (1973) studied the effects of vibration on the H reflex and demonstrated that its inhibition was more pronounced in hemiparetics than in normals. Similar experiments by Ashby and Verrier (1976) and Somerville and Ashby (1978) yielded the same results, thus implying an increase in alpha excitability due to a reduction in some central inhibitory mechanism postulated to operate at the pre-synaptic level.

Ashby and Verrier (1976) examined early hypotonic and late hypertonic patients, comparing the degree of vibration induced inhibition of monosynaptic reflexes in the triceps surae. In the flaccid patient they found :-

1) Vibration suppressed the H reflex more than in normals.

2) A normal H/M ratio.

3) A normal tendon reflex/M ratio.

4) A normal tendon reflex/H ratio.

This suggested pre-synaptic inhibition was increased on the hemiparetic side with unaltered fusimotor drive immediately after stroke.

However at the later stages of recovery they found:-

1) Less inhibition of the monosynaptic reflex by vibration.

2) A slightly increased H/M ratio.

3) An increase in the proportion of the alpha motoneuron pool activated by the tendon reflex.

Thus it appears that in the later stages of recovery there is a reduction in pre-synaptic inhibition and an increase in fusimotor drive. These results demonstrate that both the hypotheses concerning dysfunction at the spinal level can be accommodated when the total time post-CVA is taken into consideration.

The principle of reciprocal control between agonist/antagonist (Sherrington, 1906) is fundamental to

the approach adopted by Yanagisawa and his co-workers (Tanaka, 1974; Yanagisawa et al, 1976). Their concern was that stroke produces spasticity in the extensors of the lower limb concommitant with an apparent weakness in the flexors. However although the flexors appear weak, Hohmann and Goodgold (1960) demonstrated that an H reflex could be elicited in the pretibial muscles of stroke patients, but not in normals. This finding was confirmed by Tanaka (1974) and taken to indicate an exaggeration of the monosynaptic reflex in the seemingly weak muscle. Yanagisawa et al (1976) interpreted this as indicative of a spastic tendency in the pretibial muscles, but one which is swamped by the greater spasticity of the antagonistic flexors. This resulted in the hypothesis that the lesion caused by stroke results in an inbalance of reflex interaction between the opposing muscle groups.

In addition to projecting monosynaptically to alpha motoneurons, Ia afferents from the primary endings of the muscle spindle form disynaptic inhibitory connections with the alpha motoneurons of the antagonist via the Ia inhibitory interneurons (Eccles, 1969; Hultborn, 1972; Matthews, 1972) Therefore a system exists whereby the stretch of an antagonist by agonist contraction creates Ia afferent impulses which have the potential for inhibiting agonist activity. Tanaka (1974) and Yanagisawa et al (1976) studied this system by examining the reciprocal effect of stimulation of afferent fibres,

ie. the degree of inhibition of the antagonist H reflex by stimulation of the agonist afferents.

About the ankle they found a significant Ia inhibition from the extensor afferents to the flexor motoneurons, but little Ia inhibition from the flexor fibres to the extensor motoneurons. From this finding, Yanagisawa et al (1976) explain the imbalance of tone in the lower leg to be due to a release from supraspinal inhibition of Ia interneurons, with a bias in favour of the extensors.

Another inhibitory system which has been implicated in hemiparesis is recurrent inhibition (Katz and Pierrot-Deseilligny, 1982). The axons of the alpha motoneurons have branching collaterals which activate interneurons named Renshaw cells, after Renshaw (1941). In turn these bodies project back to the alpha pool in an inhibitory manner, thus completing a negative feedback loop of recurrent inhibition (Eccles et al, 1954). Renshaw cells also receive projections from the supraspinal level (Hultborn and Pierrot-Deseilligny, 1974). Additionally, the cells form inhibitory connections with the Ia inhibitory interneurons (Hultborn et al, 1971) and the gamma motoneurons linked to the muscle which the alpha motoneuron supplies.

Using a complex method of reflex conditioning, Katz

and Pierrot-Deseilligny (1982) examined to what extent Renshaw cell activity could be supraspinally modulated during a hemiparetic movement and found that normal facilitation of activity was absent. Following Hultborn et al (1979), they postulate that the supraspinal control of Renshaw cells may act as a variable gain regulator of motoneuron output and that this control is lost following stroke. Also, since there is recurrent inhibition of the Ia interneurons they additionally propose there is a dysfunction in the regulation of reciprocal inhibition.

In reviewing this area of research it is notable that the focus of study has shifted from hypotheses concerning the control of the individual muscle, to control of the agonist/antagonist linkage. However, it is difficult to draw any firm conclusions about changes in spinal functioning following stroke, partly because there are so few studies and partly because the particular investigators seem more interested in elucidating control mechanisms in general. An exception to this criticism is the work of Miller and his colleagues. Miller and Hammond (1982) attempt to give a comprehensive understanding of dysfunction in the hemiparetic at the spinal level based on :-

1) The study of spinal mechanisms underlying locomotion in the cat (Miller and van der Meché, 1975; Miller et el, 1975).

2) An electronic analog model of spinal mechanisms

(Miller and Scott, 1977).

3) The study of hemiparetic arm movements (Gandy et al, 1977; De Souza et al, 1980).

By comparing the kinematics and electromyograms of stepping in the cat, in preparations ranging from normal through decerebrate to spinal, Miller and van der Meché (1975) noted that there was a basic pattern of activity which although spinally generated, could be supraspinally modulated. This led Miller and Scott (1977) to outline a model of the spinal generation of movement in the cat with specific emphasis on the alternation between flexion and extension about a uniaxial joint. In the electronic model it was found that if background excitation to the analog alpha motoneurons and Ia interneurons was reduced, then the network became fixed in either flexion or extension. A similar result was obtained if excitation of the Ia interneurons only was reduced. However in this case, when excitation became virtually zero, there was a switch from asymmetric activation of one or other alpha pool, to activation of both groups.

A task analagous to cat locomotion on a treadmill was devised to study human arm movement (Gandy et al 1977). This involved the turning of a wheel in a stirring motion. Compared to normal subjects and the activity of the unaffected arm, the hemiparetic arm of stroke patients turned the wheel with a lower velocity,

less smoothly, and often halted around the point in the movement where the task demanded a transition from flexor to extensor activity. Examination of the EMG's at this point revealed an inability to switch off biceps activity when initiating triceps activity. This led to the conclusion that the principal deficit caused by stroke could be in the appropriate selection and execution of muscle synergies. It is proposed that underlying this is an interruption of supraspinal projections to the Ia interneurons resulting in a dysfunction of reciprocal inhibition at the spinal level.

1.3 - Treatment of hemiparesis by physiotherapy

Central to modern day physiotherapeutic treatment of stroke is the belief that the repetition of prescribed movement patterns within the constraints of certain whole body postures will promote the restitution of a functional physical independence. The origin of this essential concept can be traced to the ideas and work of Frenkel, for it is he who laid the foundations of the methods of treatment fundamental to the rehabilitation of neurological dysfunction (Licht 1973).

In 1889 Frenkel presented a paper at a congress in Germany on the treatment of tabetic ataxia. His concern was that treatment should not be based on the idea of strengthening apparently weak muscles. Rather his analysis of the condition emphasised not a loss of power, but a breakdown in sensori-motor mechanisms, resulting in a malfunction in control. Consequently, he stressed that treatment should consist of frequent repetition of active movement by the patient, during which it was supposed that cerebral registration of visual and somatic information concerning movement abnormality, would promote the learning of more correct movement production (Licht, 1973).

Frenkel's innovative ideas were sufficiently influential to cause the inauguratation of the world's first inpatient service based on the concept of

functional rehabilitation, in the Salpetrière Hospital in Paris (Licht, 1973). However, Frenkel's insights into neurological movement disorder were not adopted generally by clinicians, and by 1911 medical opinion still interpreted repeated exercise as being a method whereby repeated use of neural pathways in some way unblocked resistance to central impulses. Until the advent of World War II, the very idea that Frenkel had argued against, that exercise strengthened weak muscles, became the dominant theme of the emergent rehabilitation professions.

Much of the reason for this lay in Lovett's work (Lovett, 1917). He devised a test of the movement capacity of individual muscles in patients with poliomyelitis. A seemingly logical evolution from this, was the development of therapeutic procedures based on the training of individual muscles, for a variety of clinical conditions (Hirt, 1967). However, with the exception of Clayton (1924,cited-Westcott, 1967) the treatment of hemiplegia by active rehabilitation cannot at that time be considered as a general rule. Licht (1973) discusses:

"....faint hearted physicians who endorsed great caution in applying exercise early in any form of muscle weakness."
and follows this with:

"In the third and fourth decade of this century, the management of hemiplegia in most of the world could be characterised as inadequate"
and

"....the world literature is almost silent on hemiparetic rehabilitation during this period"

Therefore up until the 1940's rehabilitation can be considered as either:-

a) Non-existent, or

b) Based on a principle of testing and strengthening individual muscles.

According to Hirt (1967) and Licht (1973), the large number of severely disabled casualties in World War II stimulated an appraisal of this attitude. It was realised that some action needed to be taken with regard to the vast numbers of brain injured to remedy (a). Secondly, it became apparent that the goal of rehabilitative intervention, viz. rapid, optimal recovery, was not being achieved (point (b)).

It also seems that around that time there was a dawning awareness that procedures designed for the treatment of poliomyelitis were not applicable to <u>all</u> forms of paralysis. As far as therapy was concerned this awareness was initially translated into reality by Hermann Kabat and Temple Fay.

<u>Proprioceptive Neuro-muscular Facilitation</u> - In 1945 Kabat started to examine the physiological basis for the treatment of hemiplegia from the perspective of the work of Sherrington (Sherrington, 1906). His questioning led to the development of proprioceptive facilitation as a treatment method for hemiplegia (Kabat, 1950; 1952;

1961). In the development of this method Kabat, a physician, worked with two physiotherapists: Margaret Knott and Dorothy Voss. This led to the founding of a novel system of therapeutic exercise for hemiplegia and hemiparesis (Knott and Voss, 1956) known as Proprioceptive Neuromuscular Facilitation (PNF). The concepts underlying PNF are drawn almost entirely from an interpretation of the studies of reflex physiology (Sherrington, 1906; Coghill, 1929) and of normal human motor development (McGraw, 1943; Gesell and Amatruda, 1945). The philosophy behind PNF, is that the patient has a potential for improved motor function which can be exploited by understanding and treating the dysfunction (Voss, 1967).

In adopting a neuro-developmental approach, Knott and Voss (1956) evolved a stuctured treatment programme based largely on the concept of recapitulation. Following stroke it is argued that the patient has lost the ability for movement acquired during early development. The key assumption concerning development is that it consists of building a capability for complex activity out of more primitive abilities. For example, Voss (1967) claims the rolling and then crawling activities of the infant are necessary preludes to the later development of a dynamic, erect posture. Thus ,before relearning sitting or standing balance, the patient too must show competence in these more primitive

acts.

The above example also serves to illustrate a fundamental aspect of PNF therapy: the use of total movement patterns through combinations of postural reflexes and voluntary movement. The theoretical concept underlying this is that any motor act involves the entire nervous system, in the sense that movement consists of modulation of postural responses.

Since Knott and Voss (1956) claim development proceeds in a cephalocaudal/proximodistal manner, progression of movement in therapy follows this direction. In the case of the act of rolling, it is held that movement by the head and neck will elicit or reinforce trunk activity:-

"When combined with head and neck rotation, during rolling toward prone, the agonistic flexion patterns of the contralateral extremities follow the direction of the head and neck pattern, which has in itself initiated elevation of the shoulder girdle from the supporting surface. The extremities complete elevation of shoulder girdle and pelvis and proceed to rotate the trunk toward prone. The undermost extremities, toward which the head turned, have extended appropriately so as to adjust to the total movement. With reversal of direction, rolling toward supine, the antagonistic head and neck rotation pattern leads with the antagonistic extension patterns, completing the rotation of the trunk." p 846
and

"...the asymmetric tonic neck reflex with its ipsilateral movements of the extremities may be used to reinforce the total pattern of rolling...." p 844
The influence of reflexes as reinforcers to patterns of movement derives from the idea that infant motor development is dominated by reflex activity.

Performing a total movement pattern involves the structured translation from one body posture to another. Typically, as in the example above, the aim of this procedure is to rehabilitate a key movement component by other movement components. In doing this the patient is guided by the therapist. Therefore a principal role for the therapist is to manually guide and determine the initial and final body position of the patient. However the act of rehabilitating a movement component is qualified by the extent of recovery of that particular movement. It is in this context that the use of postural reflexes is exploited to the maximum:-

"Where the objective is to permit the greatest ease of performance, the patient is positioned so that the tonic labyrinthine reflexes may support his effort. Where the objective is to increase the demand on the patient's effort, he is positioned so that the influence of the tonic labyrinthine reflexes must be overcome. For example, if a patient has difficulty in initiating flexion of the hip and knee while supine (reflexly favourable for extension), he may be able to perform this in the side-lying or lateral position (reflexly favourable for flexion of the uppermost extremities), or in the prone position, with the lower extremities extending over the edge of the table, or in the creeping position (reflexly favourable for flexion of the lower extremities). By such positioning the Valsalva phenomenon may be more easily circumvented, in that effort in such positions is less demanding." page 865.

The idea that in certain stages in the treatment the patient is required to overcome a reflex tendency, introduces another fundamental aspect of PNF: maximal resistance. It is held that this process determines the degree of neural excitation delivered to the muscle. The underlying concepts to this idea are given below and are

ascribed to the work of Sherrington (1906, -cited Kabat, 1961).

1) Irradiation - the channelling of excitation from stronger to weaker muscle groups.

2) Successive induction - the idea that by alternating between opposing patterns of movement agonist response will improve.

3) Reciprocal innervation - following from (2) it is claimed that as the response of the agonist increases, inhibition of the antagonist is achieved.

Points (2) and (3) appear not only to be derived from neurophysiology, but also from neurodevelopment. Knott and Voss (1956) state that the patient alternates between phases of flexor or extensor dominance in much the same way as the developing infant. They interpret the rhythmic movements of infants as a procedure to combat dominance, and thus recommend alternation as a practice in adult therapy. Finally, during a treatment session the prescribed actions should be repeatedly attempted (the point made by Frenkel).

Complementing the instruction and handling techniques of the therapist, PNF recommends the employment of adjunctive physical agents. For example the use of ice to relax spasticity.

Neuromuscular Reflex Therapy - At the same time as Kabat, but working independently, Fay also began to consider the neurophysiological basis of therapy. His resulting

insights into the organisation of movement were similar to Kabat's, differing only perhaps in his phylogenetic or evolutionary perspective that viewed human movements as based upon the responses of lower phyla. His view was that human reflexes are elements of primitive behaviour which is more or less evident according to the degree of control exerted by more recently evolved cortical systems (Fay, 1948; 1954; 1955).

As in PNF, Fay's approach to treatment derives from developmental patterns of posture and movement, and exploits reflex behaviour. The fundamental concept underlying treatment is that it should commence with simple movements that build upon whichever reflexes are prevalent. Although over-active reflexes are considered indicative of pathology, they are not considered abnormal, but rather are seen as essential to the restitution of controlled movement. Similarly to PNF, treatment progresses from the learning of low level mobility through to higher levels of function.

Brünnstrom Therapy - In the 1950's, drawing from the work of Kabat and Fay, Signe Brünnstrom commenced the development of her own approach to treatment. Brünnstrom's initial concern was that although a database existed on the motor behaviour of stroke patients (Magnus and de Kleijn, 1912; Riddoch and Buzzard, 1921; Walshe, 1923) and could be transferred readily to the day to day practice of physical therapy, it was not. Acknowledging

the work of Twitchell (1951), she inaugurated a programme of reflex training structured around a definitive statement of expected stages of recovery:-

"Immediately following the acute episode, flaccidity is present and no movement of the limbs can be initiated (Stage 1). As recovery begins, the basic limb synergies or some of their components may appear as associated reactions, or minimal voluntary movement responses may be present. At this time spasticity begins to develop (Stage 2). Thereafter, the patient gains voluntary control of the movement synergies, although full range of all synergy components does not necessarily develop. Spasticity has further increased and may become severe (Stage 3). Then some movement combinations that do not follow the paths of either synergy are mastered, first with difficulty, then with more ease, and spasticity begins to decline (Stage 4). If progress continues, more difficult movement combinations are learned as the basic limb synergies lose their dominance over motor acts (Stage 5). With the disappearance of spasticity, individual joint movements become possible and coordination approaches normal (Stage 6). From here on, as the last recovery step, normal motor function is restored..." page 34

Brunnstrom was led to this classification of recovery by her clinical observations of around 100 patients between 1954 and 1956 and thus describes her treatment programme as being:-

"..based on the typical recovery stages of these patients, as an indication of the approximate extent of recovery in the central nervous system " page 35.

Consequently in Brünnstrom therapy, evaluation and treatment are inextricably linked.

The concepts underlying treatment are derived from Fay's (1946) phlyogenetic perspective of motor behaviour and Jackson's (1884) elucidation of the hierarchial nature of functioning of the nervous system. Brünnstrom believes that the basic limb synergies described by Twitchell (1951) are primitive spinal patterns, retained

over the course of evolution, which are normally modified and integrated into normal motor behaviour by the influence of higher centres. When supraspinal activity is occluded normal reflexes become exaggerated and pathological reflexes appear. Brünnstrom claims that since the limb synergies described by Twitchell (1951) always precede the restoration of higher control, they constitute a necessary stage in recovery which should be aided and encouraged. Treatment therefore capitalises upon the patient's reflex behaviour and can be summarised as follows:-

1) Following from the work of Riddoch and Buzzard (1921) and Walshe (1923) on associated reactions, limb synergies are elicited and reinforced by forceful contractions of the uninvolved side.

2) From studies of posture (eg Magnus and de Kleijn, 1912) the reflex mechanisms used are the tonic lumbar, neck, and labyrinthine reflexes.

3) Magnus (1924) demonstrated in animals that the same peripheral stimulus, eg tail pinching, can evoke a different motor response depending on the position of the responding part of the body. Therefore correct positioning of the patient is seen as essential during treatment.

4) Drawing from Mott and Sherrington's (1895) study of the devasting effects of sensory denervation on motor function and Hagbarth's (1952; 1960) work on skin stimulation, the use of peripheral stimulation is

emphasised as a technique for eliciting or altering motor responses.

Bobath Therapy - The developments in therapy outlined above all occurred in the U.S.A.. During the 1950's and 60's in the U.K., Karl and Bertha Bobath devised an approach to treatment which has much in common with PNF and Brünnstrom therapy:-

1) A neuro-developmental basis.

2) Principles derived from experimental reflex physiology.

3) The retraining of patterns of movement and not the strengthening of individual muscles.

4) The use of carefully planned handling and positioning techiques.

However there is a major conceptual difference. The Bobaths see the essential deficit following stroke as being the derangement of the "normal postural reflex mechanism" (Bobath and Bobath, 1964). They point out that hemiparetic spasticity is never found in isolated muscles, always in patterns. Therefore they refer to abnormal tone as being postural tone, rather than muscle tone. They view the principle disability of the patient as being spasticity. This constrains movement within limited synergic patterns and prevents isolated joint movement. Even before spasticity is visibly or palpably apparent, they believe it is latent and will appear sooner or later (Bobath, 1978). Consequently treatment is geared to prevent the entrenchment of developing

spasticity. This is in sharp contrast to Brünnstrom's approach, wherein attempts are made to encourage the synergies constituting the spastic pattern (Stages 2 and 3). In Bobath therapy, there is a positive avoidance of the reflexes and reactions which might reinforce spasticity and which Brünnstrom encourages.

Bobath (1978), follows Brünnstrom in citing the work of Magnus (1924; 1926) as evidence for the value of positioning during therapy:-

"...Magnus formulated his shunting rule which went further and which can be applied to the motor responses of more highly developed organisms. He stated that at any moment during a movement or postural change, the central nervous system mirrors faithfully the state of the body musculature. Expressed in a different way, it means that the changing state of the body musculature during movement is constantly reflected in the distribution of excitatory and inhibitory processes within the central nervous system." page 15.

However the positioning advocated by Bobath (1970) is designed to inhibit the abnormal reflexes, which constitute the spastic pattern, and redirect neural activity into more desirable patterns of activity.

Bobath also differs from PNF and Brunnstrom on another major point:-

"Working with various modalities of sensory input, ie with specific sensory stimulation such as icing, brushing, vibration or relaxation, etc., as the exponents of certain other treatments recommend, is not, in our view, the answer to the problem." page 14.

She goes on to state:-

"The patient sees and hears, he localises touch, his proprioception is normal and he perceives movements and postural changes. But notwithstanding this normal sensory input, the patient can react only with abnormal postures and movements. The reason for this is that the

lesion, in effect, 'cuts off' higher integrated activity and produces a kind of short circuit into the released abnormal patterns of spasticity." page 14.
The rationale behind Bobath therapy can therefore be summarised as follows:

"...the patient must be helped gradually to gain control over his abnormal postural reflex activity, to by-pass the short circuit into abnormal patterns, and so enable more normal patterns to become established again." page 14.

Conclusions on Physiotherapy

1) Given points (1) and (2) on page 33, there is an urgent need for the neurodevelopmental and neurophysiological basis of physiotherapy to be appraised and updated. This is essential for any treatment approach founded on research in areas which are rapidly changing and evolving. At the theoretical level physiotherapy has literally stagnated. There are potentially important basic aspects of research which need elaboration and could be influential on treatment practice. For example, the work reviewed on pages 12 - 21, on spinal reflexes, offers some support for the idea that stroke releases abnormal reflexes and for the notion that a goal for therapy should be the restitution of normal agonist/antagonist reciprocal control. However there is little evidence to support Bobath's contention that lower levels of control are divorced from higher function, and much of the theoretical basis has recently been argued as dubious (Keshner, 1981).

2) The evidence for treatment regimes is wholly based on clinical experience. One of the intentions of the above review was to demonstrate that a large database of

clinical experience exists. However the time is long overdue for aspects of that database to be translated into testable hypotheses which can be objectively assessed. The difference in opinion between Bobath (1978) and Brünnstrom (1970) on which positioning techniques to adopt, or which reflexes or reactions to encourage, reduces to a difference in subjective interpretation, rather than objective analysis, of observed clinical phenomena. Fundamental questions, such as whether one bodily position facilitates movement better than another, or indeed whether spasticity affects voluntary movement at all, remain unanswered. In short, little objective evidence exists on motor dysfunction following stroke.

3) Physiotherapy concentrates on the abnormality of movement with little reference to how normal movement is controlled. A main contention of this thesis is that by adopting the theoretical constructs and techniques utilised in the behavioural study of normal motor skills, a start can be made to answer the criticisms raised in conclusions (1) and (2).

1.4 - The study of normal motor skills

The approach to the study of movement in experimental psychology is to treat it as a behaviour and subject it to analysis. The basic idea is to examine the same movement, or compare and contrast different movements, under varying behavioural or environmental conditions. In this way hypotheses can be generated concerning the underlying control processes and the types of information necessary for their operation. Traditionally psychologists have considered movement as a motor response. For example, in an aiming movement of the hand, typical measures are: reaction time (the time it takes to prepare the response); movement time (the time it takes to execute the response); and accuracy (the amount of error in the response). However, increasingly, researchers are concerned also with the performance of movement and examine the microstructure of the movement as well.

Adams' closed-loop theory - One of the most pervasive influences in the study of motor behaviour over the past two decades has been Adams' (1971) theory of motor learning. Central to Adams' theory is the hypothesis that peripheral feedback from somatic proprioception and knowledge of results of response outcome, are crucial to the acquisition of skilled motor behaviour. Adams viewed learning to be the product of the strengthening of two hypothetical traces:-

1) The memory trace - which selects and initiates

responses.

2) The perceptual trace - which is responsible for response recognition.

During learning it is proposed that knowledge of results (KR) is used to correct errors and thus, adjust each response in relation to the previous one. In this early stage the perceptual trace , which is derived from visual and proprioceptive feedback, is built up with respect to KR rather than through a comparison of feedback over trials. As learning becomes more advanced, there is a gradual evolution of a correct and consistent response and proprioceptive information can be meaningfully compared to the perceptual trace, thus obviating the necessity of KR.

The typical learning task studied by Adams and his colleagues (Adams et al 1972) consisted of slow, self-paced, precision positioning of a lever using unidirectional and uniarticular arm movements. Adams et al (1972) tested the theory by examining the role of feedback and KR through augmenting or minimizing the subject's opportunity for feedback and providing or withdrawing KR. They demonstrated that feedback (primarily visual) determined learning and sustained skilled performance.

In Adams' theory, the efferent commands to the muscles are determined by the memory trace, with their

degree of correctness assessed by comparison of ongoing feedback with the perceptual trace. Adams' theory is thus a closed-loop theory of motor control which empnasises the importance of:-

1) Continuous visual monitoring.

2) Somatic proprioception.

Although much of Adams' theory came to be questioned on these two issues his stimulating effect on motor research is widely acknowledged. For example, Kelso (1978) acclaims it as having:-

"..led to a rebirth of an important area of psychology that lay virtually dormant in the post-war period." p 474.

The open-loop position - Adams' postulate that continuous kinesthetic or visual monitoring was necessary to skill acquisition and motor control soon came under attack from Jones (Jones, 1972; 1973; 1974a; 1974b; 1974c). Jones (1972) argued that if the CNS is capable of centrally monitoring efference, then other sources of information concerning movement were redundant. This hypothesis was largely borrowed from Helmholtz's idea, of a copy of the efference projected to the muscles, being sent to a centre where it can be matched with peripheral feedback and the movement evaluated. Jones (1972) concept of centrally monitoring efference differs in that the comparator contains a set of efference copies, which are compared with the cortico-fugal signals prior to their arrival at the periphery. Due to this central checking process, proprioceptive information is deemed to be

unnecessary.

Jones adopted this stance largely on the evidence of studies of the effect of deafferentation in animals (eg. Taub and Berman, 1968), which claimed to demonstrate that peripheral inflow is not necessary to the performance of complex patterns of movement, even in the new-born. Jones (1972) tested the central monitoring of efference hypothesis in a movement reproduction experiment using a linear positioning task similar to Adams et al (1972). This required the voluntary reproduction by blindfolded subjects of three sorts of criterion movement:-

1) Passive movement.

2) Active movement to an experimenter defined stop (Constrained movement).

3) Active movement to a target of the subjects own choosing (Preselected movement).

Jones argued that only condition (3) would allow central monitoring of efference since in (1) and (2) the subject could not know in advance the end point of the movement. He also argued that proprioceptive information was available across all the conditions. He found that replication of condition (3) was more accurate than either of the two other movements. On the basis of this, he concluded that central monitoring of motor outflow, rather than of proprioceptive inflow, is a necessary condition for accurate retention of a voluntary movement.

The linear positioning task used by Jones (1972) is typical of the experimental approach adopted in the study of motor skills in the early 1970's. At that time there was much debate as to whether movement was under open- or closed-loop control and Jones (1974) represented an extreme position within the open-loop camp. In contrast to Adams (1971), Jones suggested that visual information concerning movement is only useful in the form of KR.

Several other investigators had questioned the necessity of continuous visual monitoring. Keele and Posner (1968) trained subjects to move a fixed target distance within set times ranging from 150 to 450 milliseconds. They then examined the probability of hitting the target when the lights in the room were either on throughout the course of the movement or switched off just as the subject commenced moving. They found that only in movements lasting 250 milliseconds or more was the probability of hitting the target greater when the subject had the opportunity to continuously monitor his movement.

This led Keele and Posner (1968) to impose a 200 millisecond processing limitation on closed-loop control involving vision and Keele (1968) proposed that movements are largely under the control of preprogrammed sets of instructions sent to the muscles. Although others (eg Carlton, 1981; Lee et al, 1983) have argued that the use

of visual information can be faster, some form of open-loop control appears to operate. Further evidence for this stance came from Newell (1973) and Schmidt and Wrisberg (1973) who demonstrated that when KR is provided, visual feedback is not necessary to the learning of projectile and rapid aiming tasks. Also Faust-Adams (1975) indicated that in the linear positioning task visual feedback of criterion location is all that is necessary.

Jones' (1972) demonstration of the superiority of preselected criterion movements over constrained or passive movements was replicated by Stelmach et al (1975). However Stelmach et al (1975) employed an additional manipulation in their experiment. The central monitoring of efference hypothesis states that the efference copy against which movement is judged, is coded in terms of movement extent. Thus it predicts superiority of distance reproduction over location reproduction, since the latter demands a different efference copy to be accurate. However Stelmach et al (1975) found reproduction of the end-point of the criterion movement to be superior to reproduction of its extent. This result suggests that proprioceptive location cues (Laabs, 1973) may be crucial for accurate reproduction but does not explain the preselection effect.

In a bid to resolve this issue, Kelso (1977) compared reproduction following a constrained/passive or preselected/passive criterion movement and found it to be higher after the latter. This suggested that the crucial effect of preselection is that it supplies the system with knowledge of the goal in advance.

The mass spring model - The hypothesis that knowledge of terminal location may be an important factor in the control of movement emerged from a series of experiments by Bizzi and his colleagues (Bizzi et al, 1971; 1976; Polit and Bizzi, 1979) on head and forearm movements in the monkey. Bizzi et al (1971) compared the accuracy of normal versus loaded movements of the head. When a constant load was applied over the course of a head movement, there was an increase in EMG activity, which Bizzi et al took to indicate increased proprioceptive activity, and the target was undershot. However, as soon as the load was removed, a correction was made, and the designated target position attained. Bizzi et al concluded that this result indicated the maintenance of a program for final head position which was not affected by increased proprioceptive activity.

Sudden loading and unloading during movement, as opposed to constant loading, although perturbing the movement, did not affect eventual attainment of a target position (Bizzi et al 1976). This was the case even in

the absence of visual or vestibular cues and following deafferentation, thus supporting the hypothesis of final location programming independent of proprioceptive input.

Polit and Bizzi (1979) extended these findings in a similar series of experiments involving arm movements. Using elbow movements, the monkeys were trained to point to a target light without sight of the arm. Normal pointing was then compared with instances when a positional disturbance was applied during the reaction time to move. No difference in attainment of target position was found. However in the latter case successful attainment of the target was dependent on knowledge of the position of the arm relative to the body. When this was altered intact animals still exhibited accurate pointing, but the deafferented monkeys did not. Polit and Bizzi (1979) argued that these results demonstrated afferent information to be of value in updating or adjusting motor programs in relation to different postural sets, rather than in the execution of movement. However it should be noted that the capability of monitoring efferent (Angel,1976) or afferent (Capaday and Cooke,1981) information during movement does exist.

On the subject of what is being programmed, Bizzi and his colleagues interpreted their findings as evidence for the equilibrium point or mass spring hypothesis of motor control (Asatryan and Fel'dman, 1965; Fel'dman

1966). Under this hypothesis the agonist and antagonist about a joint are conceived as a pair of springs, the resting levels of which can be set. When the limb is stationary a state of equilibrium exists between the forces acting on the joint. The position of the limb is altered by setting a new equilibrium point and adjusting the length tension relationship of the agonist and antagonist accordingly. Thus, due to the dynamic properties of the muscles, it is argued that there is no necessity for ongoing comparison of efference with afference. (Kelso et al, 1980). The accurate performance of the deafferented monkeys, despite loading and perturbation, would seem to support this contention.

The mass spring model has received support from human studies as well (Kelso, 1977; Kelso and Holt, 1980; Cooke, 1980). Also the idea that stiffness, rather than muscle length is the controlled variable, has been found physiologically attractive (Houk, 1979). Although the theoretical structure of the model is still undergoing development and analysis, unfortunately enthusiastic proponents of the idea (eg Kelso et al, 1980) tend to elevate it to the level of ultimate explanation. Kelso and Holt (1980) examined the effects of perturbation on the accuracy of finger movements after functional deafferentation by a wrist cuff in blindfolded subjects. Compared to performance without perturbation, variable and constant error were significantly higher. However on

the basis that the differences were "very modest", they dismissed this result and claimed equifinality of movement (ie programming with respect to target position rather than a target distance).

Kelso and Holt's results could be interpreted as indicating that their subjects could not finely correct their end-point error because visual and proprioceptive information were not available. The discovery of a speed accuracy trade-off in movement control (Fitts, 1954) implied that whilst the majority of the movement is under open-loop control, fine end-point correction is under closed-loop control. Greene's (1972) ballpark hypothesis also emphasised this control distinction: pre-programming can only get the limb into the region or "ballpark" of the target, thereafter visual guidance serves to precisely home it in (Carlton, 1981). There is therefore a need for the mass spring proponents to define exactly what they mean by "equifinality".

Bernstein's two problems - Most of the experimental work in the 1970's examined simple movements, principally about one joint. Since this was thought to involve the contraction of only a few muscles, most of the hypotheses about control assumed that commands at the cortical level were identical to those received by the motoneurons, ie. there existed a direct command link between centre and periphery which contained all the necessary information for controlling the intended movement.

The publication in 1967 of the translated papers of the Soviet physiologist Bernstein, made an impact on the study of motor control which still reverberates throughout the field today (Whiting, 1983). He outlined (at least) two problems which any theory of motor control had to account for:-

1) The degrees of freedom problem.

2) The problem that the desired outcome of an act could be obtained in a variety of ways.

These problems are interrelated.

The first refers to the fact that the skeletal joints can move in more than one direction. For example, the elbow can either flex or extend and thus has one degree of freedom. There are however, other more complex joints with more than one degree of freedom (eg the shoulder). Therefore over the entire skeletal system there are hundreds of degrees of freedom and thus the potential for thousands of different patterns of movement. However, Bernstein noted that in complex movements the number of degrees of freedom controlling the action was much less than the potential of the system. Therefore if individual programs control individual muscles or agonist/antagonist linkages, not only would there be immense storage problems, but also the question arises as to how these programs interact to give co-ordinated movement.

Problem (2) is best illustrated by an often cited example. If one writes the letter "a" on a piece of paper with a pencil, or on a blackboard with a piece of chalk, the outcome is the same although entirely different sets of muscles have been used in the two instances. This means that any program for action at the higher level must contain some abstract definition of movement outcome rather than as Keele (1968) suggested, a pre-structured set of muscle commands. Bernstein's insights were partly responsible for a reappraisal by programming theorists, which led to the idea of a generalised motor program (Keele and Summers, 1976) or schema (Schmidt, 1976, (after Head (1920) and Bartlett (1932))) operating at the cortical level. However this still does not explain how an abstract idea for action is translated into actual movement and how the degrees of freedom problem might be solved.

Co-ordinative structures and tuning - A start has been made on resolving these theoretical issues by Turvey and his colleagues (Turvey, 1977; Turvey et al, 1978; Kugler et al, 1980; Kelso et al, 1980). In doing so they draw heavily on an essay by Easton (1972) and on the work of students of Bernstein (Gelfand et al, 1971; Gelfand and Tsetlin, 1971; Gurfinkel et al, 1971; Kots et al, 1971; Kots, 1977). Gelfand et al (1971) proposed that instead of commands from higher centres projecting directly to the muscles, this information is used to control the modes of interaction between lower centres, which

themselves are capable of producing patterns of movement in a relatively autonomous fashion. Evidence for the existence of such centres can be derived from studies of locomotion in deafferented or spinal preparations which demonstrate the existence of movement generators at the spinal level (Grillner, 1975; Shik and Orlovsky, 1976). Thus these centres relieve the higher levels of control from the burden of many degrees of freedom and permit programming at that level to be abstract, in the sense that it does not necessitate detailed knowledge of lower level activity. At the peripheral level this hypothesis implies that muscles are not controlled individually, but in groups. One way in which muscles are grouped is by reflexes. These consist of complex configurations of movement, often spanning many joints and involving many muscles (eg. the tonic neck and labyrinthine reflexes). Furthermore Easton (1972) suggested that through supraspinal control, reflexes can be ordered, summed or fragmented into functional units which he termed "co-ordinative structures". Under this postulate, controlled movement is envisaged as the fitting together of structures which each deal relatively autonomously with a limited aspect of the control problem.

The working hypothesis of Bernstein's students was that the spinal cord does not act as a passive agent passing on instructions from higher centres (Gurfinkel et al, 1971). This hypothesis was generated by indications

that direct supraspinal projection to the alpha motoneurons is the exception rather than the rule. Rather, the majority of descending tracts terminate at the interneuronal level (Kots, 1977). As was noted earlier in this Chapter (page 18) interneurons such as the Ia inhibitory interneurons determine the balance of excitation and inhibition across the agonist/antagonist linkage. In doing so they do not merely relay higher commands, rather their activity is determined by afferent as well as efferent activity.

A major point which Kots (1977) makes, is that supraspinal modulation of interneuronal systems occurs prior to agonist activation and biases the spinal system towards the intended action. This conclusion was derived from experimental work using techniques similar to those outlined on page 16. These studied changes in the spinal apparatus during the reaction time to initiate a voluntary movement. For example, Kots and Zhukov (1971) found that in dorsiflexion of the foot there is a biasing of the balance of reciprocal inhibition to counteract the future possibility of stretch induced contraction of the plantar flexors, before the motoneurons of the agonist are fired.

Findings such as this led to a distinction of at least two mechanisms in neural control: tuning and activation. The former anticipates and facilitates the

consequences of the latter. Tuning is not merely restricted to the agonist/antagonist pair, though (Gurfinkel et al, 1971). For example, Nashner and Cordo (1981) have demonstrated postural adjustments prior to movement in muscles quite remote from the intended agonists.

Turvey (1977) proposed that through the formation of co-ordinative structures, the system establishes which muscles will effect movement, and that through the process of tuning, the resultant movement is controlled. It is during tuning that afferent information plays a most important role. As Polit and Bizzi (1979) demonstrated, proprioceptive information is necessary at this stage to tailor efferent commands to the postural set of the body. Since actions normally involve the attainment of environmental goals, visual information is held as essential to tuning. Under this conception, the role of vision is to control movement in a feedforward mode rather than to supply feedback or knowledge of results.

Gibson's theory of ecological optics (Gibson, 1966; 1979) details the environmental potential for vision in the control of action by considering the ways in which the environment structures light. Before light reaches the eye it has been multiply reflected by the surfaces in the environment to form a complex network of rays.

Gibson calls this "ambient light" structured in an "optic array". Since the various surfaces in the environment have differing textures, sizes and angles of reflectance, the manner in which they reflect light also differs. The structure in the optic array is thus specific to the surface layout of the surrounding environment. Gibson points out that surface texture is arranged in elements. An element is conceived as being the smallest detectable difference in wavelength or intensity. This forms a contour within which there is homogeneity of reflected light and across which there is discontinuity with neighbouring elements. The concept of discontinuity is extended to the corners and edges of surfaces, such that the contours of surfaces and therefore the relationship between surfaces is specified by the ambient light.

By the principles of projective geometry, the structure in the light is lawfully and orderly translated on to the retina, such that it receives information which is invariant with respect to environmental layout. Normally there are movements in the environment and of the observer. Therefore the optic array is in a constant state of flux. However this flux is not random, but is a specification of whatever changes are occurring in the layout of the environment, due to external or observer-generated movement. Lee (1978) identified three classes of information available through vision for the control of action :-

1) Exterospecific - information concerning the actions of external objects.

2) Propriospecific - information about the relationship between parts of the observer's body.

3) Expropriospecific - information on the relationship between the observer, or some part of him, and the contents of the environment.

As the observer moves forward through the environment a global expansion pattern of texture elements is projected on to the retina through the nodal point of the lens of the eye. When moving backwards, the reverse occurs in the optic flow pattern projected to the retina: the texture elements flow away from the observer. Lishman and Lee (1973) and Lee and Lishman (1975) mimicked these changes in a study on the control of balance. Subjects stood inside a room which was suspended from the ceiling of a larger room, and which could therefore be moved forwards and backwards. When the room was moved toward the subject it produced an optical expansion pattern, specifying forward motion, which caused the subject to sway backwards. As the room moved away from the subject and created a diminishing pattern of flow, the stationary subject perceived himself to be swaying backwards and leant forward in correction. Despite the presence of vestibular and proprioceptive cues stating that he was actually stationary, the subject swayed backwards and forwards in time with the movement

of the room. Other examples of the powerful propriospecific function of vision in the control of posture and balance have been demonstrated by Dichgans et al (1972; 1976) and Lestienne et al (1979).

Any bounded patch of texture in the environment, which is not part of the observer's body, specifies an object. If this patch is moving, and if the observer wishes to interact with the object, for example in catching it, the transformation in the optic array caused by the motion of the object, additionally affords expropriospecific information. The expropriospecific function of vision has recently been ascertained by Lee et al (1983) in a task requiring precise timing of actions. The subject's task was to leap and punch a ball falling directly above him. The results indicated that the changes in elbow and knee angle necessary to accomplishing the task were governed by the time remaining before contact with the ball. This time-to-contact information was visually provided by the rate of dilation of the image of the ball in the optic array as it approached the subject.

Similar visually based time-to-contact information has also been shown to control the timing of muscle activity in falling in man (Dietz and Noth, 1978; Greenwood and Hopkins, 1980) and in leaping in the monkey (Dyre-Poulsen et al, 1980) in preparation for landing.

It would appear that a major function of vision is to predictively control action. In a simple, discrete action, such as extending the arm to press a switch, once having provided information for tuning the movement, vision enters into the act only in the final stages to home the finger onto the switch. There is therefore an economy of visual intervention in motor control. However, observation by the author of the treatment of patients at a local rehabilitation hospital, indicated that a principal consequence of stroke is that this economy is lost, as the patients rely heavily on visual monitoring of performance. This observation is reinforced by statements in the literature:-

1) Twitchell (1951) only regards recovery as complete when movement can be conducted without visual monitoring.

2) Some assessment scales, eg. Lincoln and Leadbitter (1979), have successful completion of non-visually guided tasks as one of their terminal items.

3) Continuous visual guidance of action is stressed during treatment (Bobath, 1978; Brünnstrom, 1970).

This observation was largely responsible for determining the focus of study in this thesis.

## 1.5 - Statement of the problem and aims of the thesis

This review demonstrates that little objective data exists on how the control of voluntary movement is disordered following stroke. It is argued that this lack of knowledge is partly responsible for a management of the disorder using treatment approaches founded on:-

1) Clinical observation.

2) Subjective interpretation of research which often does not specifically refer to hemiparesis per se.

The section on normal motor skills outlines hypotheses and techniques which may be profitably applied to the study of abnormal motor behaviour. Therefore the global aim of this thesis is to give a preliminary description and understanding of motor control in the stroke patient from a perspective of normal motor functioning.

In attempting to do this, the control of affected arm movement in the patient is compared and contrasted with unaffected arm and normal arm performance. Of particular interest is the role played by vision, therefore the fundamental question posed in this thesis is:-

Is visual guidance of movement of benefit to the control of arm movement in the stroke patient and if so, how does vision function?

Two complementary questions also run through the experiments to follow:-

1) Given the use of whole body positioning and movement patterning in therapy (as described by Bobath (1978)), how do the affected and unaffected sides communicate with each other? That is to say: can the neural activity in one limb influence the processes controlling the actions of the other limb?

2) Given the diametrically opposed views on managing spasticity during rehabilitation (Bobath, 1978; Brunnstrom, 1970), the issue as to whether there is any tendency for spasticity to interfere with the execution of voluntary movement, is also examined.

# CHAPTER II

## Visual Control of Repetitive Reaching Tasks

## Introduction

The three experiments reported in this Chapter represent a preliminary attempt at understanding how the hemiparetic uses visual information for movement control. Two broad questions are being posed here. Firstly, is the large investment of visual attention to motor activity observed in stroke patients of therapeutic benefit, and secondly, if so, then how and by what means? A dotting or pointing task was used. This paradigm has been widely used as a tool for the study of many aspects of visuo-motor control ever since its introduction by Woodworth (1899). Basically it consists of matching the end position of the hand to a spatially located target.

The general approach here is a comparative study of extent and direction of end point errors over time when affected, unaffected and normal arms reach to a target in the transverse plane under differing visual and target conditions. The required movement is midline extension of the arm, as diagrammed in Figure 2.1.1, with performance depending upon controlled coupling of fixed wrist position, elbow extension and shoulder adduction and internal rotation. Following the discussion of normal perceptuo-motor performance given in the previous Chapter, a general conclusion can be reached.

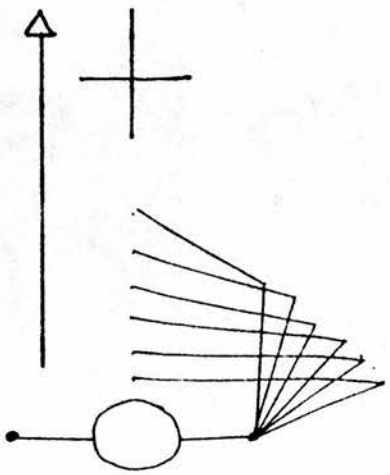


Figure 2.1.1 Diagram of the task movement.

Arrow indicates direction of movement - Midline Extension. Multiple images of stick arm indicate the action requires controlled coupling of the elbow and shoulder.

In carrying out a simple discrete action , such as the above, vision is necessary in the tuning of the movement to establish in which direction the target lies, how far away it is, and its size, whilst also furnishing data on the position of the effector in relation to the target and the rest of the body. However, during the execution phase visual influence is negligible, entering the control process only at the end stages of the movement when fine corrections may need to be made. Errors made by normal subjects in this task under impoverished visual conditions would be expected to be minimal. With hemiparetic subjects this might not be the case. If it is true, as suggested by the observations of Chapter One, that hemiparetics are dependent on visual control then errors would be expected to be large in the absence of vision.

By comparing extent of error across conditions which differentially manipulate particular aspects of visual information, an idea of the nature of visual control employed by patients can be gained. For example, is it important to have arm and target continually available in the field of vision, or would sight of one or other alone be sufficient to approximate to a standard of performance achieved under conditions of full vision? With normals, Stubbs (1976) reported that if target location alone remains visible, then there is little decrement in performance compared to a condition where there is no

manipulation of visual information. He reasons that this is to be expected since the position of the normal limb is known through somatic proprioception. One of the possible effects of stroke can be loss of somatic proprioception from the mechanoreceptors (Johnstone, 1978). Typically therapists attribute the necessity of continuous sight of the affected limb action as a substitute for sensory or proprioceptive loss. Yet even without this, there is a probability that Harris' (1971) concept of "inappropriception" could apply in describing the quality of somatic information available for motor control in these patients. This could result in hemiparetic aiming performance, in a target alone condition, being very poor indeed.

Lee (1978) suggested that in activities involving a long series of discrete movements, e.g. typing, although continuous visual guidance is not needed and indeed not desirable, vision occasionally enters into the action as an overseer to tune-up or re-calibrate somatic proprioception. This is because kinesthetic information from the body is subject to drift and thus deviates in its accuracy as a source of information. Paillard and Brouchon (1974) demonstrated this phenomenon in one of a series of experiments designed to study position sense in normal man. Their apparatus consisted of two parallel tracks mounted vertically. The subject's left index finger was positioned either actively or passively on one

track and the task was for the subject to line up his right index finger with it. They found that if there was a time delay of over 8 seconds between positioning the left hand and attempting to line up the right, accuracy was significantly reduced in both positioning conditions.

With this in mind, the experiments reported below were designed with the trials for a given condition presented without interruption, in a block, in the course of which a uniform setting of experimental variables was maintained throughout. In typical aiming tasks, e.g. Beggs and Howarth (1970), the experimental situation is, during inter-trial intervals, essentially neutral to the variables being manipulated. The procedure adopted here was conceived as a control for possible drift. In the normal case, there is no reason why proprioceptive drift should be in any particular direction, except perhaps where the subject becomes fatigued. However if Bobath's hypothesis of developing spasticity is correct, then it might be expected that responses of the hemiparetic limb would increasingly deviate in the direction of flexion as illustrated in Figures 2.1.2 (a) and (b). All the movements performed in the following experiments involve primarily the elbow joint with the triceps as the prime mover. It would not be unreasonable to predict that if drift sets in over time, its pattern would be dictated by any spastic tendency, resulting in successive responses following a temporal gradient of error as shown in Figure

2.1.2.

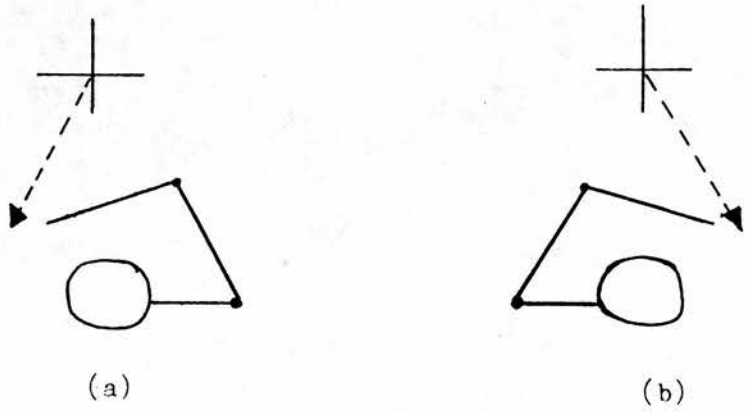



Figure 2.1.2 Predicted drift from accuracy for movements of a) the right arm and b) the left arm.

## Experiment 2.1 - Repetitive reaching to proprioceptive targets

### Introduction

The objective of this experiment was to examine the proprioceptive function of vision and its interaction with somatic proprioception. The task entailed locating with the upper limb, a target point on the lower limb. Since at all times during the experiment the subject's legs were occluded from view, the target points can be considered to be defined solely by somatic proprioception. In the task then three potential sources of proprioceptive information were available: somatic concerning knee position, somatic about the arm, and visual about the arm. Following the work of Paillard and Brouchon (1974), it was thought that in subjects with a normal, well integrated somatic proprioceptive system, accuracy under blindfold conditions would be high, but would deteriorate over repetitive trials. The reaching performance of stroke patients could be dependent upon any one of three predicted effects of the cerebral vascular accident (CVA):-

1) That it produced a random dysfunction of communication within one half of the body only.

2) That it re-arranged unilateral communication uniformly

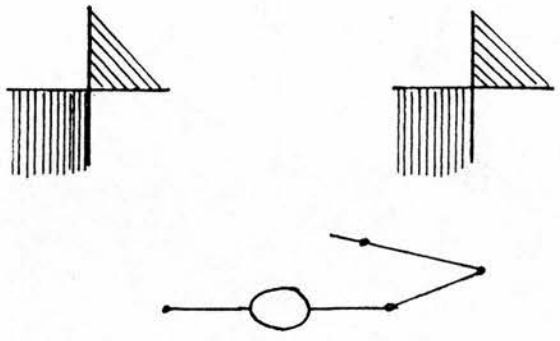
through the tendency to spasticity.

3) That the unaffected side had also been somewhat disturbed rendering proprioceptive information generally unreliable.

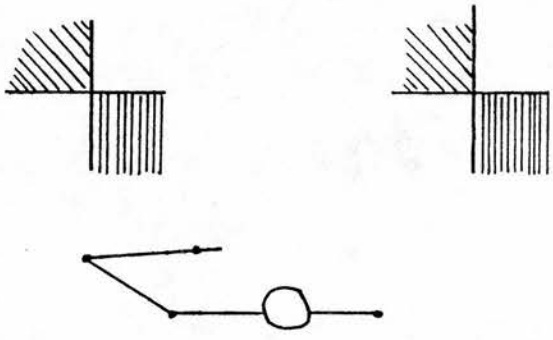
To test these predictions all four combinations of arm and knee were performed. From (1) it would be expected that there would be a gradation of quality of performance from ipsilateral action of the affected limb (worst) through both contralateral movements (equal) to ipsilateral on the unaffected side (best). (2) would predict both ipsilateral movements being more accurate than their contralateral counterparts and (3) would suggest little difference across all four combinations.

With the additional provision of visual information concerning the performing limb it was expected that accuracy would increase since one of the sources of somatic drift would be corrected for.

Following the scheme of Figure 2.1.2, it would also be expected that the end point distribution for movements of the affected limb would be uneven, according to the patterns in Figure 2.1.3. With an enhanced knowledge of the arm's position in space relative to the body through vision this distribution might not ensue and a more random pattern could emerge.



A — RIGHT HANDED MOVEMENT



B — LEFT HANDED MOVEMENT

Figure 2.1.3. Showing the relationsip of arms and targets in Experiment 2.1. Shaded quadrants represent regions of most likely and least likely directional error for the affected arm following Figure 2.1.2.

## Method

### Subjects

Six normal and six hemiparetic subjects took part in this experiment. The normal subjects were either staff or student members of the Department of Psychology, consisting of four males and two females, all being right handed. Hemiparetic subjects were selected from in- and out-patient populations of Astley Ainslie Hospital, Edinburgh. All had been diagnosed hemiparetic due to a CVA. Selection proceeded in the following manner. The experimental task was explained to the physiotherapists in both the in- and out-patient departments of the hospital and they were asked to suggest suitable subjects. Criteria for suitability were:-

1) Ability to perform the arm movements necessary to the task.

2) A willingness or interest in participating.

3) No gross spatial neglect.

4) No gross sensory loss.

5) No comprehensional difficulties.

6) Right handedness.

Criteria (3), (4) and (5) were assessed from the patient's medical notes.

Once the patient had agreed to take part, his medical notes were consulted to check for factors apart

Table 2.1.1
Subject details

| SUBJECT | SEX | AGE (YRS) | HEMISPHERE OF LESION | ARM * FUNCTION | TIME POST CVA (WKS) |
|---|---|---|---|---|---|
| H1 | M | 55 | L | Poor grip, good elbow and shoulder function | 12 |
| H2 | M | 62 | L | Poor wrist and finger function, good shoulder and moderate elbow function. | 6 |
| H3 | F | 58 | L | Good all round. | 9 |
| H4 | M | 59 | L | Good all round. | 10 |
| H5 | M | 60 | R | Good grip and elbow function, poor shoulder movement. | 8 |
| H6 | M | 65 | R | Good grip, poor elbow and shoulder function. | 6 |

* Obtained from the patients' therapy notes.

from the stroke which might affect his perceptuo-motor performance e.g. a visual field defect or arthritic complications in the joints of the arm. If any such disorders were found then the patient was not used as a subject. The patient was then seen by the experimenter, the nature of the task explained and the patient's ability to carry it out to experimental requirements assessed. If the patient could perform the task as shown, without fatigue, he was finally selected as a subject. Details of hemiparetic subjects are outlined in Table 2.1.1

The description of each subject's affected arm function was obtained from the physiotherapist treating him. Function of the other arm was said to be normal in all cases.

Design

This consisted of three levels of conditions of mixed design: Visual x Target x Arm (2x2x3)

The visual conditions were:-

NV - The subject was blindfolded.

V - The subject was permitted to see the movement but not the target he was aiming for

The target conditions were:-

IK - Isilateral knee

CK - Contralateral knee

The arm conditions were:-

AA - Affected arm of the stroke patient.

GA - Good arm of the stroke patient.

NA - The right arm of the normal subject.

Apparatus

The apparatus and experimental set-up was as shown in Figure 2.1.4. The bottom board was backed with inch thick foam rubber. This served the two-fold purpose of firmly anchoring the board over the subject's knees and of preventing tactile localisation of the knee. The front and lateral sides of this board were marked out and numbered every 5mm in order that the position of the target knee relative to this board could be calculated. A top board was fitted over the bottom one by means of two strips of wood fixed to its underside. These strips were the width of the bottom board apart and acted as runners in the X-direction. This enabled the top board to be slid in the x direction, without slippage in the Y-direction whilst the bottom board remained stationary.

On the top board was a large sheet of graph paper, the sides of which were marked off as co-ordinates in the same manner as the bottom board. The front of the top board was marked off at 5cm intervals as shown in Figure 2.1.4(b), so that it could be both accurately centered and repositioned in the x direction relative to the bottom board dowel markers. The reason for the sliding property of the top board was to enable alteration of its position relative to the bottom board during trials. A footstool was provided to elevate the knees with respect

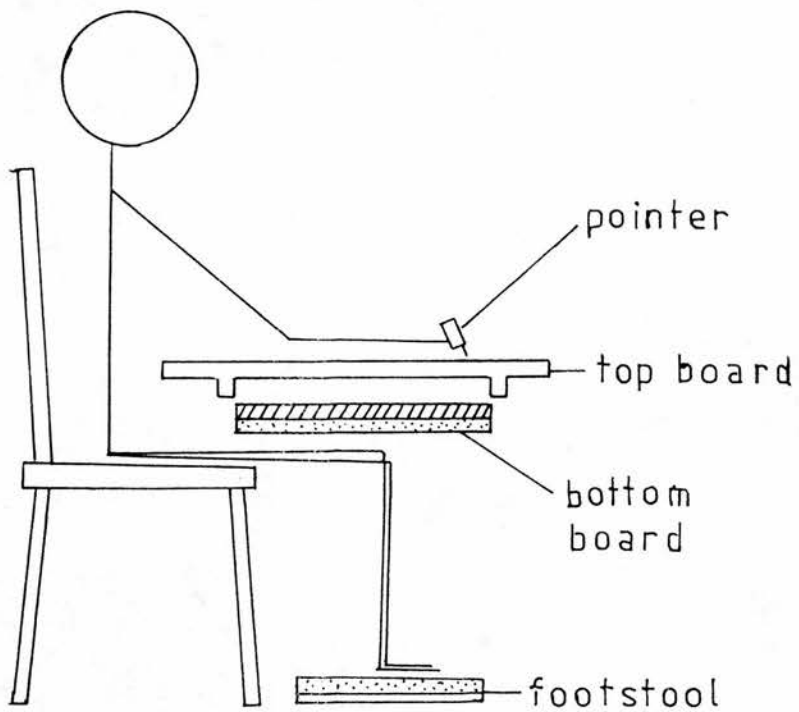



Figure 2.1.4 A Experimental set-up.

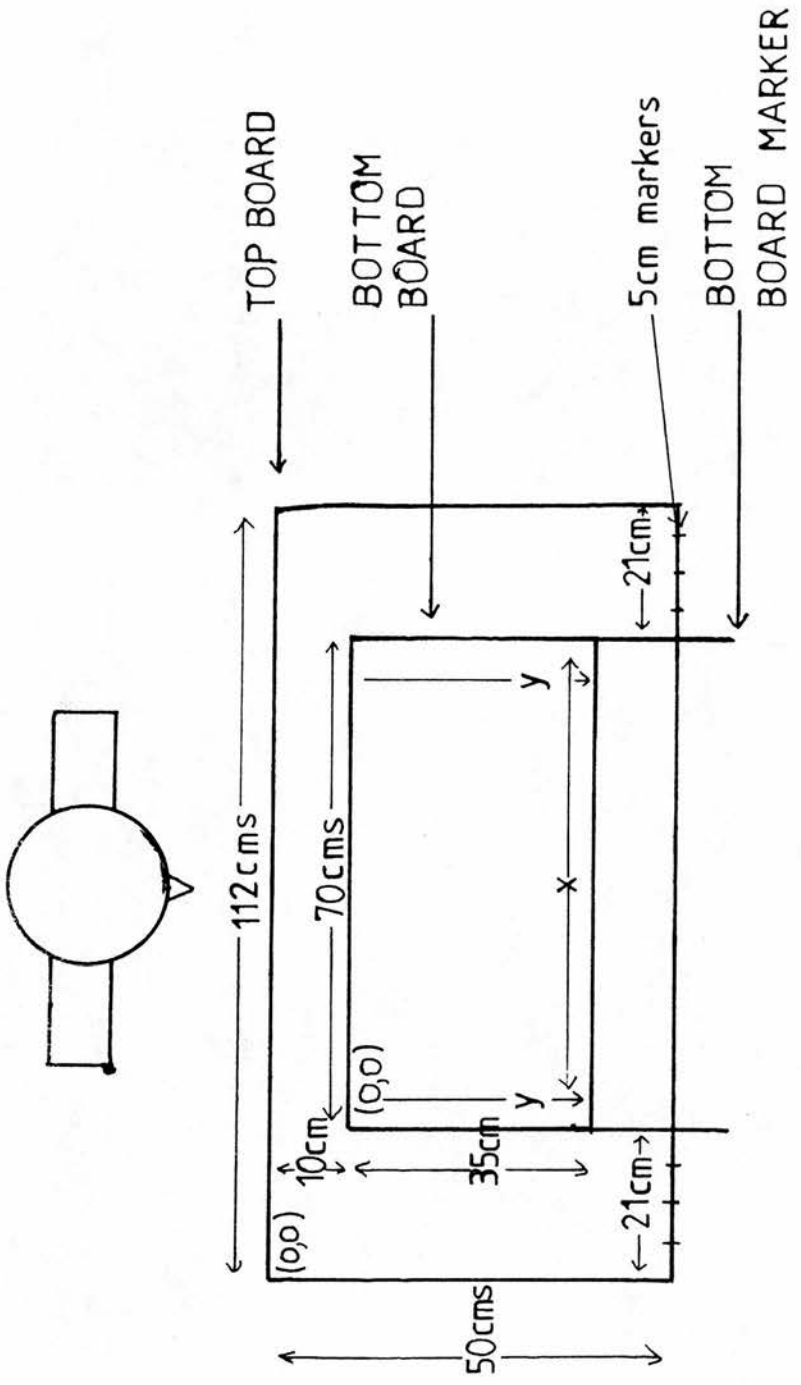



Figure 2.1.4 B Relationship of top and bottom boards.

to the pelvis. This was surfaced with 2 inch thick foam rubber and aided in anchoring the feet.

Pilot work had demonstrated the difficulty many patients have in normal pointing with the forefinger extended in isolation. Therefore, for the purposes of obtaining accurate measurement of pointing responses a short, wooden dowel, 1 inch in diameter, with a panel pin protruding from one end, was available. It was found that all hemiparetic subjects could use this pointer quite comfortably. A blindfold was used in the no vision condition.

Procedure

For normal subjects the experiment was conducted in an experimental cubicle at the Department of Psychology. The hemiparetic subjects were seen in a treatment room of the hospital's Physiotherapy Department. Pilot work had shown tnat the patients tended to tire quickly and become bored, therefore sessions were kept to a maximum of thirty minutes. Due to slowness of movement, time to set up, practice trials and rest periods, usually only two blocks of trials could be carried out per person, per day. Due to this and also the necessity of sometimes having to re-run blocks of trials, e.g. because of interruptions, the time period for the entire experiment was anything up to two weeks. To balance normal subjects for this, their experimental period extended over one week.

At the first session the subject was introduced to the apparatus and the task was explained to him. He was then asked to locate the target point of the knee (just behind the patella) a total of thirty times, with the pointer starting from a hand position at the midline of the trunk. This was done for all four hand knee combinations with hemiparetic subjects, and with the right hand to both knees with normals. Performance was observed so that accuracy over the required number of reaches under conditions of full vision could be assessed.

Over the period of the experiment, conditions were presented to each subject in a random order. Each condition consisted of twenty seven trials. This figure was chosen for two reasons. Firstly, pilot work had demonstrated that about thirty consecutive movements at any one time was a fair estimation for a fatigue cut-off point for most stroke patients. Secondly, twenty seven balanced precisely the number of times each of the nine positions of the top board relative to the bottom, could be used. Before commencing each block of trials, the subject was allowed practice under full visual conditions until he felt comfortable about the task. His feet were then placed on the footstool and its height adjusted as necessary. The particulars of the condition were stated with the relevant knee and arm indicated manually as well as verbally. It was stressed that he should keep his

body, apart from the arm, immobile over the course of the trials and that he should not attempt to reproduce a previous trial.

The subject was then instructed to assume the starting position. He was told only to move on the command of "Now" to where he thought the target position to be, to touch the board with the pointer and then return to the starting position and await the next "Now" command. In the V condition he was told that he must keep his eyes closed. On hearing "Now" he was required to open them before moving, keep them open during the movement and only close them again when leaving the board to return to the target starting position.

The boards were then placed on the subjects thighs. The position of the target knee was recorded using the co-ordinates of the bottom board. If it was a NV condition the blindfold was put on. The trials then commenced. After each movement the end location of the pointer was noted from the co-ordinate system of the top board, before the command to initiate the next movement was issued. In V conditions the top board was moved randomly to a different one of the nine possible positions, after the end location had been noted and before the trial commenced. Over the twenty seven trials each position was used three times. Error of measurement was estimated at +/- 0.125cm for all the experiments in

this Chapter.

Throughout, the experimenter changed his recording position since in pilot trials one subject reported using his voice as a positional cue to keep responses consistent. Subjects were warned of this beforehand. Immobility of the body was carefully monitored. If the subject moved seating or leg position, the block was abandoned. If the subject attempted to aid forward motion of the arm by bending at the waist he was reminded not to do so, the trial ignored and an additional one done. When this persisted, it was interpreted as fatigue and the block of trials repeated at some later stage. Once completed, the apparatus was removed and the subject was allowed to rest for as long as he liked.

## Results

### Scalar Error

Mean scalar error (r) was calculated for each condition using $\bar{r} = \sum\sqrt{x^2 + y^2}/n$ with the target point being defined as the origin. Individual means and group means and standard deviations are presented in Table 2.1.2. Group data is also illustrated in Figure 2.1.5. Three ANOVA's were performed on the group data:-

1) A three way repeated measures ANOVA, 2x2x2, (Arm x Vision x Target) compares the affected arm with the unaffected.

2) A mixed ANOVA with the unaffected arm and the normal arm as groups and 2 x 2 levels of Vision x Target, compares the unaffected with the normal arm.

3) To examine the effects of the Vision and Target conditions on the normal arm, a 2 x 2 repeated measures ANOVA was performed.

There were no significant effects or interactions in the first of these comparisons. As revealed in Figure 2.1.5, this indicates little difference in overall accuracy of performance between the affected and unaffected arms and no discrimination across the experimental conditions. Figure 2.1.5 shows that the performance of normal subjects is more accurate than that of CVA patients with either affected or unaffected arms.

Table 2.1.2
Mean Scalar Error (cm)

NORMAL SUBJECTS

| CONDITION | N1 | N2 | N3 | N4 | N5 | N6 | GROUP |
|---|---|---|---|---|---|---|---|
| IA>IK NV | 10.55 | 1.50 | 5.89 | 5.98 | 5.32 | 0.98 | 5.04 (3.99) |
| IA>CK | 8.22 | 7.27 | 3.26 | 2.70 | 8.10 | 5.36 | 5.82 (2.93) |
| IA>IK V | 5.66 | 3.22 | 8.45 | 3.20 | 8.12 | 3.50 | 5.36 (2.45) |
| IA>CK | 2.99 | 7.20 | 6.29 | 9.63 | 5.70 | 6.40 | 6.37 (2.16) |

HEMIPARETIC SUBJECTS

| CONDITION | H1 | H2 | H3 | H4 | H5 | H6 | GROUP |
|---|---|---|---|---|---|---|---|
| AA>AK NV | 9.40 | 10.38 | 3.21 | 9.90 | 13.49 | 13.27 | 10.11 (3.65) |
| AA>UK | 9.68 | 3.23 | 6.49 | 12.17 | 10.13 | 10.62 | 8.72 (3.28) |
| AA>AK V | 10.57 | 4.18 | 5.70 | 3.62 | 12.11 | 24.56 | 10.13 (7.87) |
| AA>UK | 6.40 | 10.58 | 6.90 | 6.34 | 5.11 | 17.96 | 8.88 (4.82) |
| UA>UK NV | 2.27 | 6.24 | 1.79 | 10.33 | 22.17 | 16.67 | 9.83 (8.28) |
| UA>AK | 8.20 | 10.00 | 16.04 | 6.83 | 12.99 | 12.06 | 11.09 (3.40) |
| UA>UK V | 9.24 | 16.38 | 3.80 | 5.88 | 17.61 | 13.00 | 10.89 (5.62) |
| UA>AK | 8.10 | 6.82 | 8.76 | 2.75 | 5.15 | 37.72 | 11.55 (13.00) |

GROUP - tne group mean with the standard deviation in brackets.
CONDITION - for explanation of abbreviations refer to page72.
> - denotes reaching to.

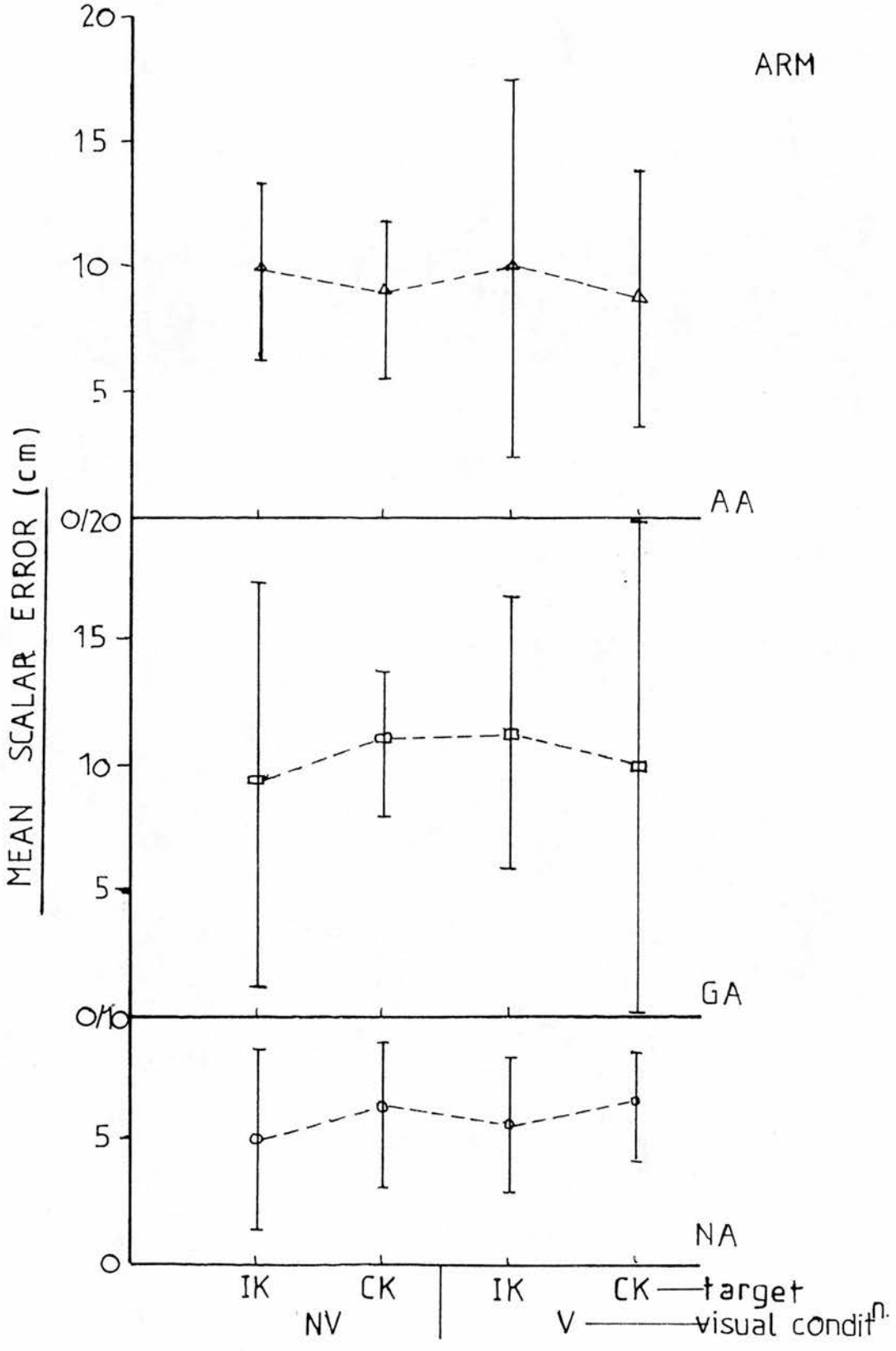



Figure 2.1.5. Mean Scalar Error with S.D. across for conditions of Experiment 2.1.

Table 2.1.3
RMS Error (cm)

NORMAL SUBJECTS

| CONDITION | N1 | N2 | N3 | N4 | N5 | N6 | GROUP |
|---|---|---|---|---|---|---|---|
| IA>IK NV | 1.16 | 2.42 | 2.00 | 2.20 | 1.81 | 2.42 | 2.08 (0.32) |
| IA>CK | 2.60 | 1.60 | 1.56 | 2.14 | 1.90 | 2.60 | 2.07 (0.46) |
| IA>IK V | 2.63 | 10.35 | 1.43 | 2.43 | 1.98 | 3.65 | 2.62 (0.90) |
| IA>CK | 1.57 | 2.29 | 2.35 | 2.63 | 2.54 | 4.28 | 3.74 (3.32) |

HEMIPARETIC SUBJECTS

| CONDITION | H1 | H2 | H3 | H4 | H5 | H6 | GROUP |
|---|---|---|---|---|---|---|---|
| AA>AK NV | 1.53 | 1.58 | 2.76 | 2.70 | 7.47 | 8.08 | 4.02 (2.96) |
| AA>UK | 1.52 | 3.95 | 2.58 | 3.90 | 7.13 | 6.87 | 4.33 (2.26) |
| AA>AK V | 1.43 | 3.15 | 2.92 | 3.77 | 5.55 | 6.56 | 3.90 (2.87) |
| AA>UK | 2.53 | 1.21 | 1.80 | 1.94 | 4.66 | 17.31 | 4.91 (6.19) |
| UA>UK NV | 1.92 | 2.14 | 3.83 | 2.40 | 10.06 | 4.99 | 4.14 (3.08) |
| UA>AK | 1.62 | 2.39 | 5.49 | 4.72 | 3.50 | 8.11 | 4.31 (2.35) |
| UA>UK V | 2.25 | 2.46 | 2.42 | 3.09 | 5.40 | 7.84 | 3.91 (2.26) |
| UA>AK | 1.92 | 1.23 | 2.70 | 2.60 | 8.63 | 12.86 | 5.00 (4.69) |

Refer to the caption following Table 2.1.2.

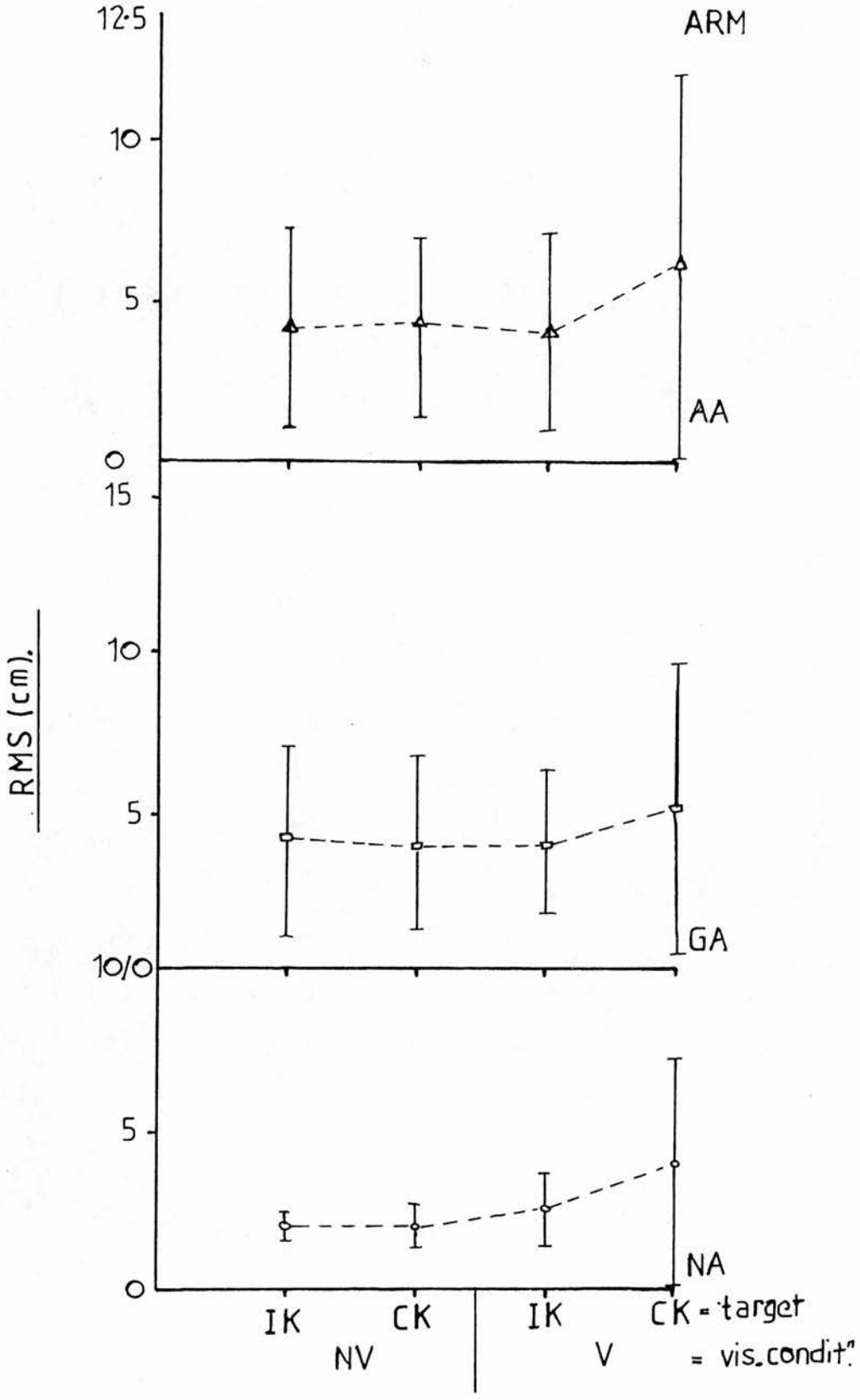



Figure 2.1.6 Mean RMS with S.D. across subjects for conditions of Experiment 2.1.

ANOVA (2) found the difference between the unaffected and normal arms to be significant ($F(1,10) = 5.45$, $p < 0.05$). Examination of normal performance alone revealed no significant differences between the experimental conditions.

Root Mean Square Error (RMS)

As a measure of consistency in deviation from target, RMS was calculated using $RMS=\sqrt{\frac{\sum_{i=1}^{n}(x_i - \bar{x})^2 + (y_i - \bar{y})^2}{n}}$ with $x_i$ and $y_i$ being the trial (x,y) coordinates and $\bar{x}$ and $\bar{y}$ being the mean (x,y) cordinate across the trials. Figure 2.1.6 shows the group means and standard deviations with individual and group values presented in Table 2.1.3. The above three ANOVA's were conducted and no significant effects or interactions whatsoever were found.

Direction of Deviation from Target

When individual trials for each subject in each condition were plotted , no consistent directional drift away from target could be discerned in any case. One subject's raw data from one condition is plotted in Figure 2.1.7 to illustrate the random nature of drift over trials.

Figure 2.1.8 represents plots of mean (x,y) points for each subject in each condition. In order to examine any directional unevenness in mean accuracy across subjects, each mean point was scored for presence or absence in a particular quadrant. Left arm data was then directionally adjusted to be right hand by reversing the sign of the x value and the results are presented in

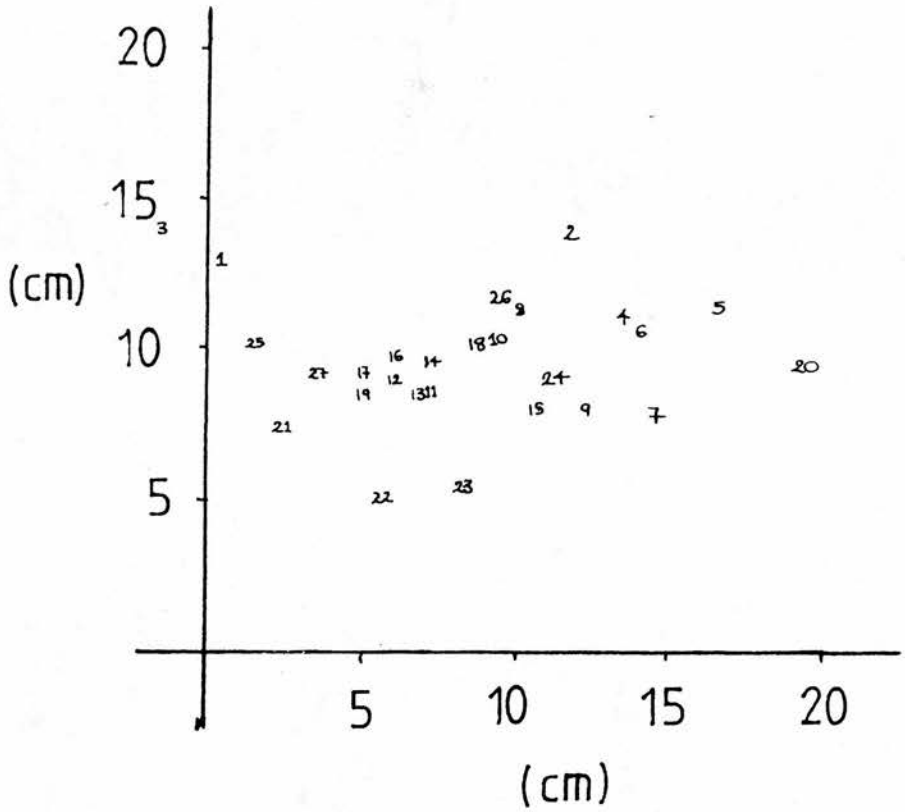
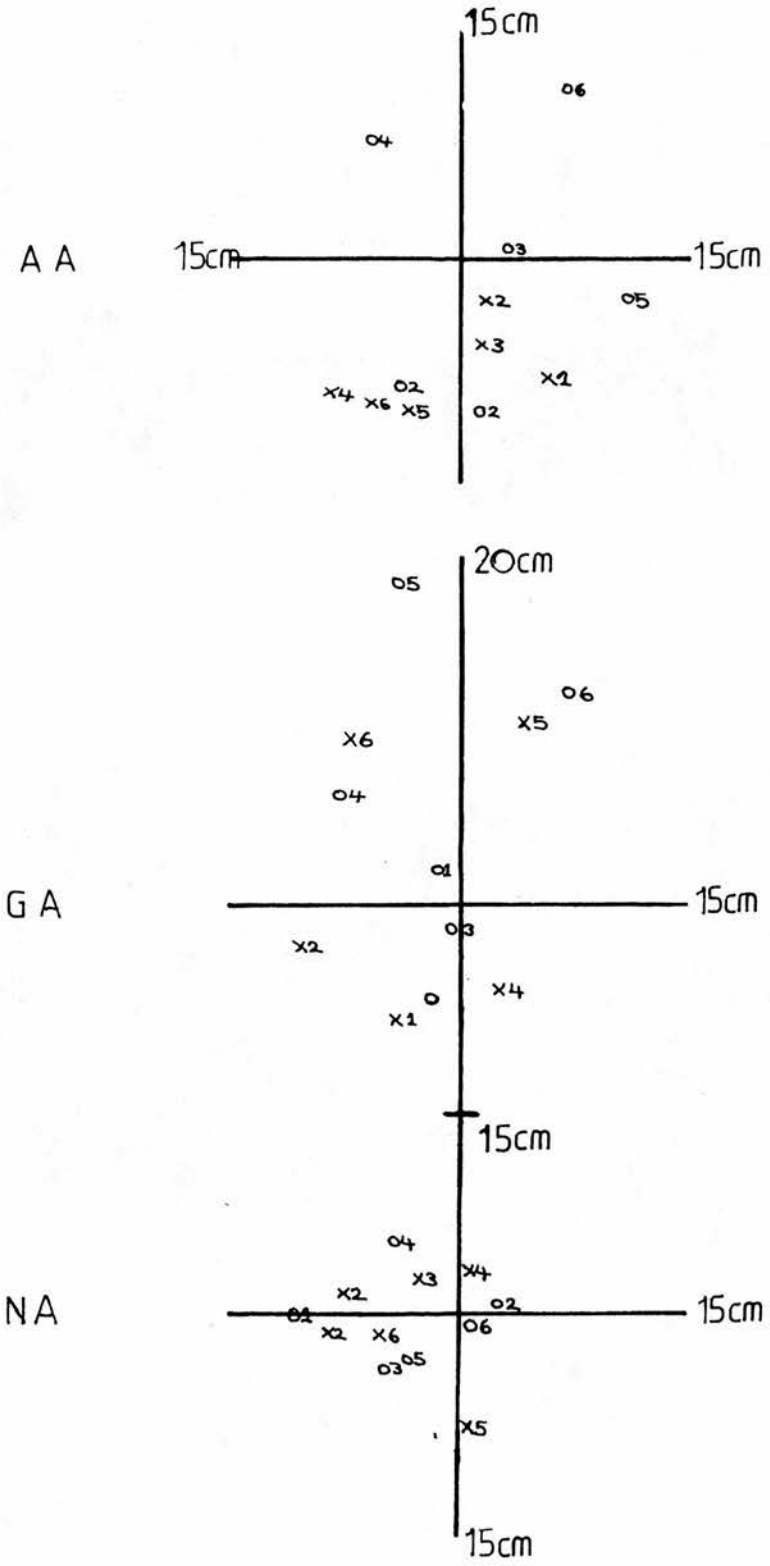
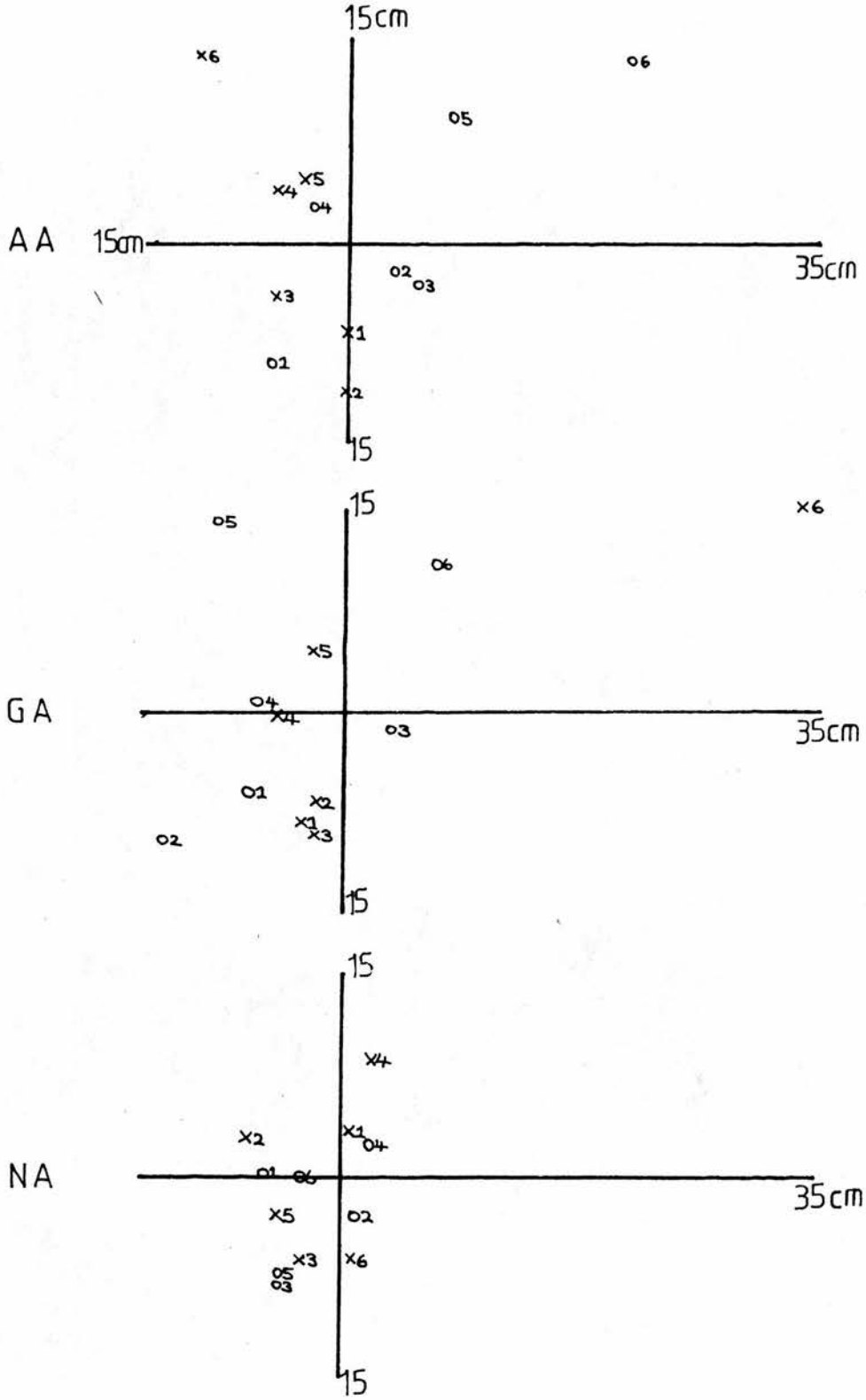



Figure 2.1.7. - Showing random pattern of drift across individual trials 1 to 27. Subject H5 reaching to the affected knee with the affected arm.




O - Ipsilateral target.
X - Contralateral target.

Figure 2.1.8. Distribution of subjects mean points in Experiment 2.1 with vision unoccluded.




O - Ipsilateral target.
X - Contralateral target.

Figure 2.1.8. Distribution of subjects mean points in Experiment 2.1 with vision occluded.

| DISTRIBUTION | | ARM |
|---|---|---|
| 8 | 3 | NA |
| 7 | 6 | |
| 4 | 8 | GA |
| 2 | 10 | |
| 6 | 3 | AA |
| 7 | 8 | |

FIGURE 2.1.9. NUMERICAL SUMMARY OF FIGS 2.1.7. WITH LEFT HANDED POINTS ADJUSTED TO RIGHT HANDED

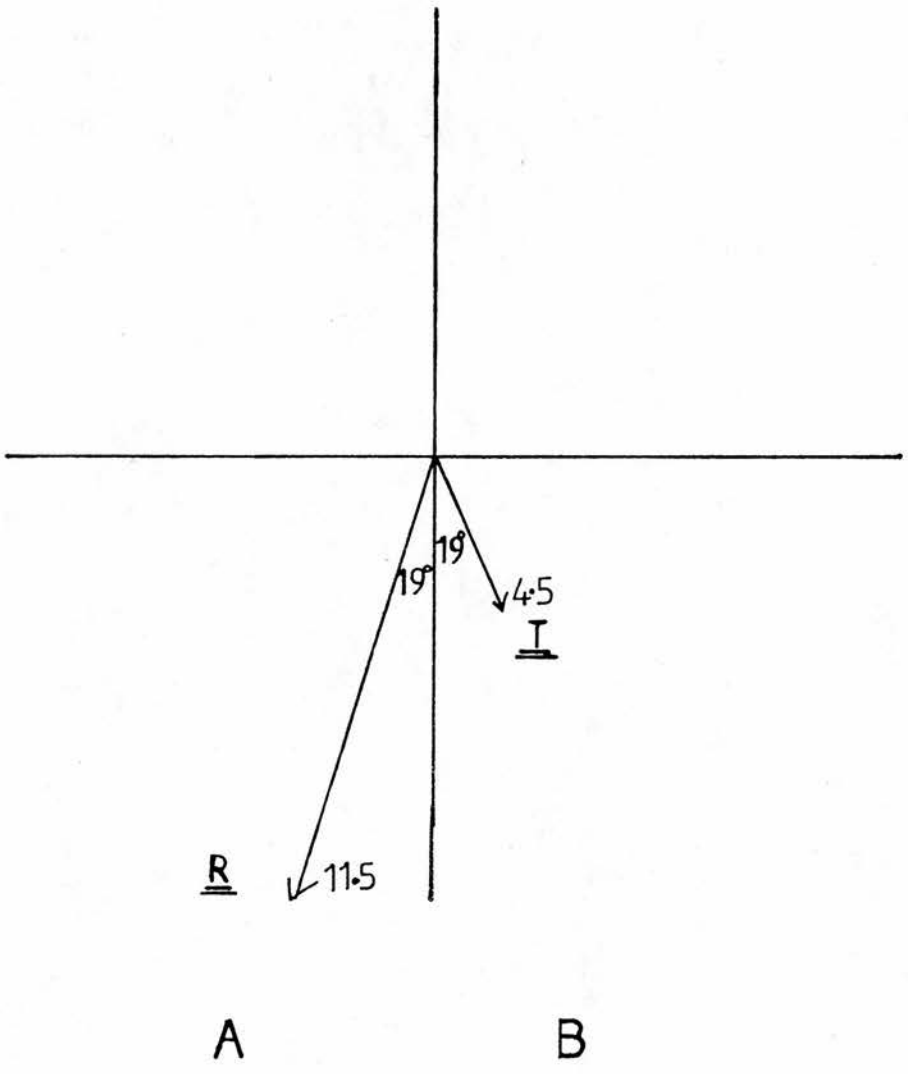



Figure 2.1 10. Vector analysis of the effect of hemiparesis on target position (T) and reaching (R).

T : Felt Knee position

R : Felt Arm position

Figure 2.1.9. A chi - square test for frequency of mean response in each of the four quadrants revealed no significant unevenness in response distribution for all three arm conditions.

The above analysis is based on an objective definition of the target point. Since the target was proprioceptive and remained unseen across both visual conditions, it may have been that the subjects somatically perceived it to be in a different position from where it actually was. In order to examine this and to assess the effect of hemiparesis upon both the reaching performance of the arm and perception of the location of the knee, the following analysis was conducted.

The group (x,y) means were obtained for three of the Arm/Target conditions for both visual conditions:-

1) The unaffected arm pointing to the unaffected knee (UA/UK).

2) The unaffected arm pointing to the affected knee (UA/AK).

3) The affected arm pointing to the unaffected knee (AA/UK).

As above, in calculating these values, left arm results had the sign of the x value reversed. The mean of UA/UK under NV (2.34, 6.41) was taken to indicate the best non-visually guided estimate of where the unaffected knee

was positioned. The mean of AA/UK was then taken as a measure of hemiparetic performance under NV to this same target. If the UA/UK mean is then made the origin, i.e. the subjective, as opposed to objective target, then the extent and deviation of nemiparetic reaching can be obtained from the difference between UA/UK and AA/UK. This yields a coordinate of (-1.07, -10.75) giving a vector of 11.34 cms in length with a deviation of 18.6 degrees clockwise from the negative half of the y axis (Figure 2.1.10A).

As above, by making UA/UK under NV the origin, comparison with UA/AK under NV is proposed as providing an indication of the effect of hemiparesis upon perceived location of a proprioceptive target. The resultant coordinate is (1.52, -4.38) giving a vector of 4.63 cms long with a deviation of 19.2 degrees anticlockwise from the negative half of the y axis (Figure 2.1.10B).

Similar calculations with respect to UA/UK (3.14, 1.50) for AA/UK (0.51, 0.21) and UA/AK (7.04,-0.32), all under V, were performed to assess deviation of the limbs under visual guidance. For the effect of hemiparesis on reaching and target perception, this resulted in vectors 63.9 degrees clockwise of the negative y axis (2.93 cms in length) and 65.1 degrees anti-clockwise of the negative y axis (4.30 cms long), respectively.

Discussion

Perhaps the most surprising result of this experiment is that it provides little evidence for improved affected arm accuracy when sight of the arm is permitted. On the face of it, this seems to contradict the hypotheses advanced at the beginning of this Chapter, concerning the possible facilitatory role of vision. The only support for this comes from the the vector analysis presented in Figure 2.1.9. The size of the vector is quite dramatically reduced when sight of the affected arm is allowed. However the vectors pertaining to unaffected arm performance remain unchanged across the visual conditions. When the experimental situation is considered this result is not surprising.

Since in none of the conditions could the target be seen, visual information was solely proprioceptive in nature, affording control for arm position with respect to the upper half of the body. Control with respect to the target rested on knowledge of the felt position of the knee. On the assumption that proprioception is normal in the unaffected arm the availability of visual information during the trials might not be expected to improve performance. However in the control of the affected arm it appears to serve a useful proprioceptive function.

As well as considering the size of the vectors,

their direction is also of interest. Although no consistent drift across trials was detected, the effect of hemiparesis on affected arm reaching is consistent in its direction with the prediction that the arm would err due to over-adduction at the shoulder and under-extension at the elbow. Underlying this could be an nder-estimation of the amount of extension necessary to counter the development of spasticity in the flexors. If this is the case then a similar underestimation could follow from the effect of hemiparesis on the felt position of the affected knee. In the lower limb, spasticity develops in the extensors, therefore the leg could be perceived as less abducted and extended than it really is. This is consistent with the direction of the unaffected arm vectors which show precisely this under-estimation.

In this experiment, the reliance on felt position for target location probably largely contributed to its failure to separate out the hypotheses advanced in the Introduction, at the objective level of analysis. Normal, as well as stroke subjects, reported that the padding into which the knee indented, increased uncertainty about a part of their body, the non-visual location of which they felt somewhat uncertain about anyway. This level of uncertainty could have swamped any changes in accuracy resulting from the experimental manipulations.

## Experiment 2.2 - Repetitive reaching to proprioceptive and exteroceptive targets

### Introduction

The main aims of this experiment were similar to those of Experiment 2.1.: (a) to determine to what extent accurate localisation of a target over time, depends upon visual monitoring of the limb; (b) to compare the two halves of the body as sources of somatic, proprioceptively defined target positions. There are several criticisms that can be made of Experiment 2.1 and these were taken into account in the design of this experiment:-

1) Arbitrarily defining a point on the knee as the proprioceptive target could have led to uncertainty about target position. In this experiment, a more obvious and punctate point is used: the tip of the index finger.

2) Previously all the targets were haptically defined. Here exteroceptive targets are also used, to provide a yardstick for any proprioceptive effects.

3) In half of the first conditions, the target was across the midline from the starting position of the arm. As one subject demonstrated, this may prove problematic to patients with right hemisphere lesions (H6, Table 2.1.2, Figure 2.1.7), therefore, in this experiment none of the

conditions demanded midline crossing.

4) Accuracy under full visual conditions was only observed. In the current experiment, accuracy is recorded in the same manner as the experimental conditions.

## Method

### Subjects

The same six normal subjects who took part in Experiment 2.1 also participated here using the same arm as before. Hemiparetic subjects were also identical except for H4. In this experiment H4 was a sixty one year old woman with a right hemisphere lesion. When she took part eight weeks had elapsed since her stroke. Her arm function was good apart from poor shoulder movement. The same selection procedure was employed in choosing her.

### Apparatus

The target cross was presented on the surface of a table. Table height was adjustable. The table top measured 50 cm square and two interchangeable surfaces were used. One was cut from wood and the other a sheet of clear perspex. In both cases the target point was 20 cm out from the middle of the subject's end of the table. The task was to reach under the table in an attempt to line up underneath the target. The exteroceptive target was a 2 cm cross. In proprioceptive target conditions this was covered with a small piece of plasticine upon which the subject placed the tip of the index finger of the arm contralateral to the intended effector.

The perspex top was used in one condition to assess performance under full visual monitoring. The opaque top

was used in all other conditions in conjunction with a cloak which could be attached to the table. This completely occluded sight of the reaching arm whilst retaining vision of the target. In conditions requiring additional removal of sight of target, a blindfold was provided.

Renewable undersurfaces were attached to the table top for recording purposes. These were sheets of stiff, white paper for the opaque surface and clear acetate sheets for the perspex surface. Recording of end-point positions was carried out by the experimenter using six differently coloured pens. The subject pointed with the dowel described in Experiment 2.1. Only in the condition of full vision could the subject observe the recording of the end-points.

Design

There were three levels of conditions in a mixed design:-

Visual Conditions - FV - Reaching was performed without any occlusion of vision.

V - The subject could see the target, but not his arm.

NV - The subject was blindfold.

Target Conditions - C - The cross or exteroceptive target.

F - The finger or proprioceptive target. Either felt or seen according to pairing with

the

visual conditions.

Arm Conditions - These were as Experiment 2.1. The locations for the experiment, time constraints, activity limits, familiarisation and practice routines, were similar to those of Experiment 2.1. Conditions were randomly ordered with each composed of thirty trials.

Once the subject was seated the table height was adjusted to his requirements and its position was such that the target aligned with the body midline. The subject was then informed of the condition and the necessary apparatus put into place. He was told that he was required to move the arm from the starting position at the edge of the table, under the table and then try and line the dowel up underneath the target and touch the undersurface. It was stressed that contact should be punctate and that bending from the waist should not be used as a supplementary force to the reach.

The experimenter then crouched beneath the table and the trials commenced. When the subject made contact with the recording surface it left a small indentation, which as the arm returned to the start, was marked by the experimenter using one of the pens. For each consecutive group of five trials a different colour of pen was used. Once thirty trials had been reached the recording surface was removed and the subject was permitted to rest.

## Results

### Scalar Error

Mean scalar error was calculated using the equation of Experiment 2.1. Individual means, group means and standard deviations are presented in Table 2.2.1, with the group data graphed in Figure 2.2.1. As in the previous experiment three ANOVA's were performed on the group data.

Comparing affected with unaffected arm accuracy, $F(1,5) = 2.22$, $p = 0.19$, shows no main effect of Arm. There are however, main effects of Target ($F(1,5) = 19.87$, $p < 0.01$) and Vision ($F(1,5) = 11.08$, $p < 0.05$). Although there are no significant interactions, Figure 2.2.1 snows sight of the target has a greater effect in improving accuracy of the unaffected arm.

The type of target has a different, but non-significant effect across the two arms. In the case of the affected arm, the proprioceptive target improves performance under NV, but under similar conditions accuracy of the unaffected arm slightly deteriorates. This latter point is also evidenced in the comparison of the unaffected arm with the normal arm data. There is a main effect of Arm ($F(1,10) = 5.67$, $p < 0.05$) and also a three way interaction ($F(1,10) = 6.69$, $p<0.05$) which post - hoc analysis reveals is due to the differential effect

Table 2.2.1
Mean Scalar Error (cm)

NORMAL SUBJECTS

| CONDITION | N1 | N2 | N3 | N4 | N5 | N6 | GROUP |
|---|---|---|---|---|---|---|---|
| NV/C | 2.41 | 8.21 | 6.87 | 4.63 | 3.24 | 3.77 | 4.35 (2.50) |
| V/C | 2.00 | 2.35 | 0.63 | 1.59 | 2.97 | 1.70 | 1.88 (0.78) |
| NV/F | 0.79 | 1.82 | 2.66 | 0.39 | 2.47 | 3.00 | 1.86 (1.06) |
| V/F | 0.36 | 1.04 | 2.71 | 0.61 | 2.67 | 3.41 | 1.80 (1.78) |

HEMIPARETIC SUBJECTS

| CONDITION | H1 | H2 | H3 | H4 | H5 | H6 | GROUP |
|---|---|---|---|---|---|---|---|
| NV/C | 1.95 | 10.92 | 6.39 | 14.03 | 11.09 | 6.16 | 8.43 (4.38) |
| V/C | 1.72 | 4.35 | 5.70 | 12.86 | 10.40 | 3.61 | 6.44 (4.29) |
| AA NV/F | 0.89 | 3.83 | 4.35 | 13.81 | 11.77 | 9.85 | 7.42 (5.11) |
| V/F | 2.38 | 3.68 | 5.86 | 10.85 | 11.15 | 3.33 | 6.21 (3.89) |
| NV/C | 4.28 | 4.92 | 4.34 | 4.27 | 15.71 | 5.74 | 6.54 (4.53) |
| V/C | 4.18 | 3.89 | 2.56 | 4.41 | 4.93 | 2.68 | 3.78 (0.96) |
| UA NV/F | 5.39 | 9.60 | 5.31 | 6.81 | 14.83 | 1.13 | 7.18 (4.65) |
| V/F | 1.66 | 3.70 | 1.02 | 2.82 | 2.38 | 3.59 | 2.53 (1.06) |

GROUP - tne group mean with the standard deviation in brackets underneath.
CONDITION - for explanation of abbreviations see page 97.
> - denotes reaching to.

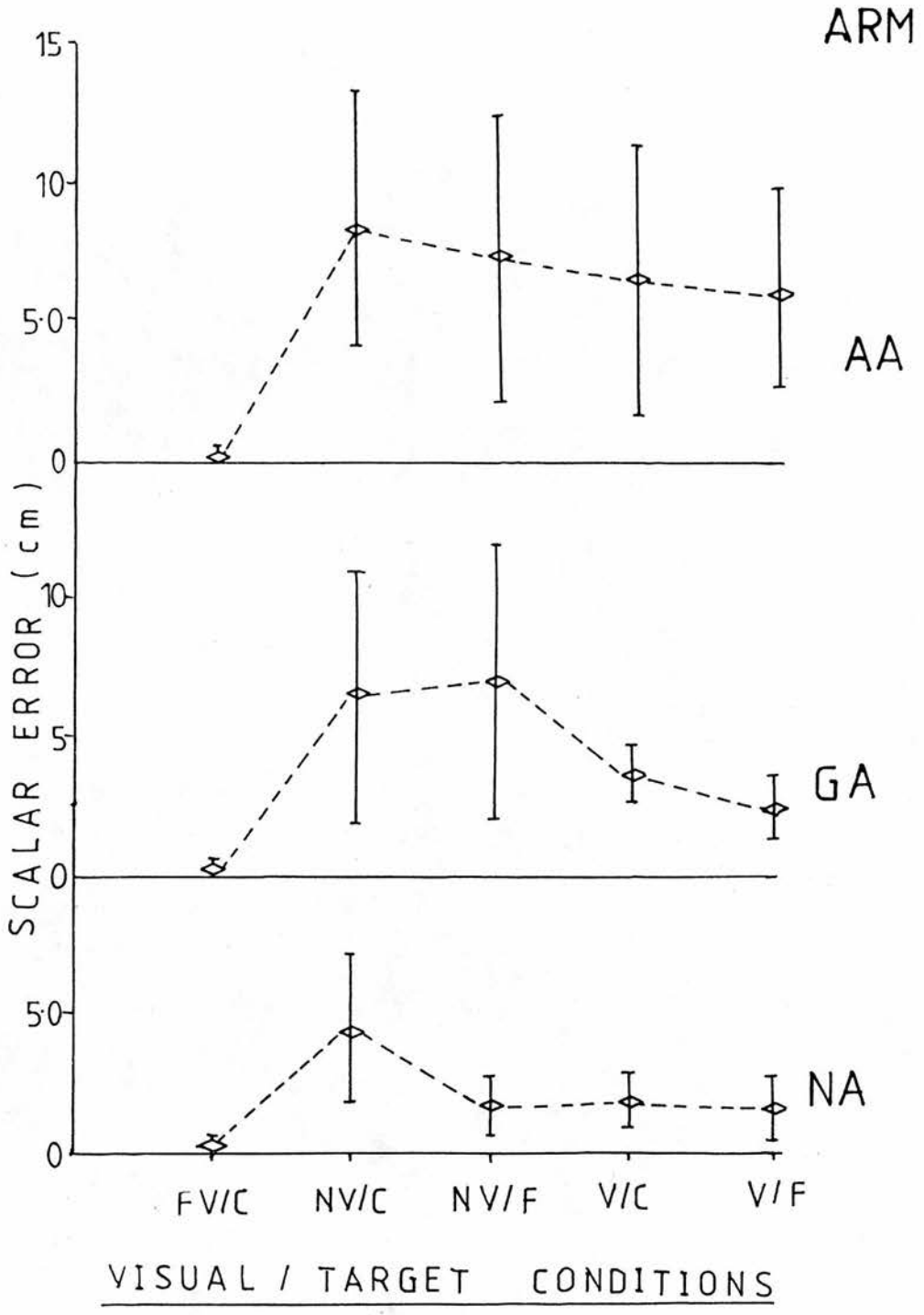



Figure 2.2.1. Average and standard deviation of mean scalar error across subjects for Experiment 2.2

Table 2.2.2
RMS Error (cm)

NORMAL SUBJECTS

| CONDITION | N1 | N2 | N3 | N4 | N5 | N6 | GROUP |
|---|---|---|---|---|---|---|---|
| NV/C | 1.06 | 3.77 | 2.51 | 1.08 | 2.02 | 1.46 | 1.98 (1.04) |
| V/C | 0.97 | 1.13 | 0.98 | 1.43 | 1.39 | 1.67 | 1.26 (0.28) |
| NV/F | 0.95 | 1.23 | 1.10 | 1.80 | 1.80 | 1.90 | 1.96 (0.42) |
| V/F | 0.82 | 1.02 | 1.04 | 1.17 | 1.27 | 1.79 | 1.68 (0.33) |

HEMIPARETIC SUBJECTS

| CONDITION | H1 | H2 | H3 | H4 | H5 | H6 | GROUP |
|---|---|---|---|---|---|---|---|
| NV/C | 2.42 | 4.50 | 3.24 | 7.06 | 4.30 | 3.07 | 4.10 (1.65) |
| V/C | 2.42 | 1.23 | 2.02 | 3.12 | 3.71 | 1.28 | 2.30 (1.00) |
| AA NV/F | 1.87 | 1.54 | 1.63 | 5.23 | 3.59 | 2.13 | 2.67 (1.98) |
| V/F | 1.81 | 1.55 | 2.69 | 3.55 | 2.72 | 1.25 | 2.25 (0.87) |
| NV/C | 2.57 | 2.13 | 1.59 | 4.91 | 3.42 | 2.24 | 2.81 (1.19) |
| V/C | 1.40 | 1.12 | 0.98 | 1.58 | 2.05 | 1.43 | 1.43 (0.38) |
| UA NV/F | 1.49 | 2.31 | 2.56 | 4.71 | 6.07 | 1.65 | 3.13 (1.85) |
| V/F | 1.05 | 1.03 | 1.44 | 1.29 | 1.90 | 1.63 | 1.39 (0.34) |

Refer to Table 2.2.1 for explanation of abbreviations.

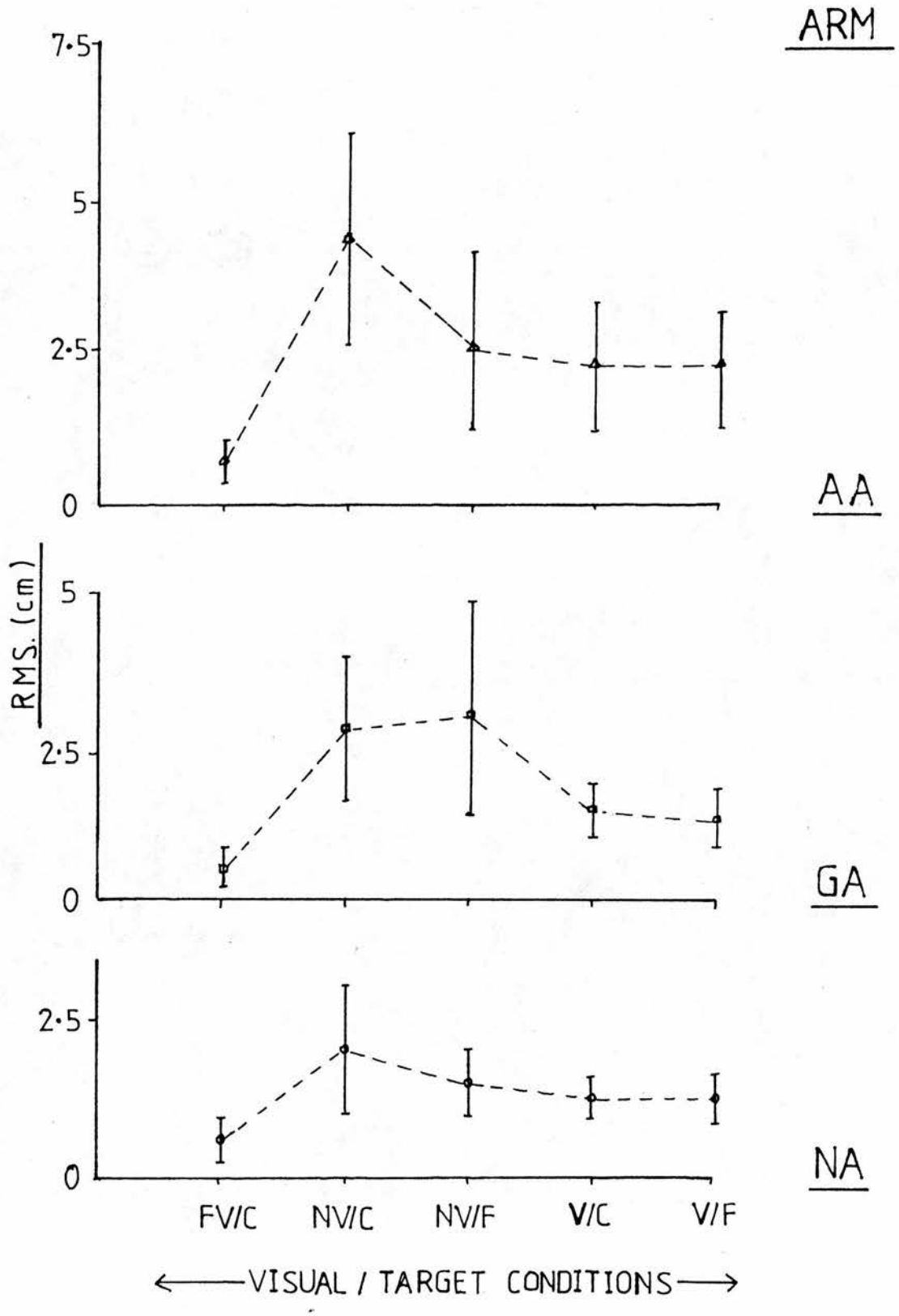



Figure 2.2.2. Mean R.M.S.

tne proprioceptive target has under NV. Although in general tne unaffected arm is less accurate than the normal, tnis difference is only significant for this combination of conditions.

Analysis of the normal arm errors shows a main effect of Vision ($F(1,5) = 7.20$, $p < 0.05$) coupled witn a significant two way interaction ($F(1,5) = 8.29$, $p < 0.05$). This indicates that, unlike the performance of the unaffected arm of a paretic patient, when information concerning target position is provided by body mechanoreceptors under NV, accuracy of normal subjects is as high as when sight of target location is permitted.

Root Mean Square Error

This was calculated for each condition following the procedure of Experiment 2.1. Individual means, group means and standard deviations are tabulated in Table 2.2.2 with the group means and standard deviations illustrated in Figure 2.2.2. Identical ANOVA procedures to those performed on Scalar Error were conducted on this measure.

On comparison of the affected and unaffected arms there is a main effect of Arm, $F(1,5) = 8.83$, $p < 0.05$, with tne unaffected arm having the lower RMS. However this is qualified by a significant Arm x Target interaction ($F(1,5) = 6.78$, $p < 0.05$) due to RMS dropping

for the affected arm when the target is proprioceptive under NV, but rising for the unaffected arm under the same conditions.

There is a main effect of Vision ($F(1,5) = 10.56$, $p < 0.05$) with the reduction in RMS, brought about by sight of the target, more pronounced for the unaffected arm. This point is reinforced from the other two ANOVA's. There are no significant differences across conditions when the normal arm is examined alone. Comparing the normal with the unaffected, there is no difference. However there is a main effect of Vision ($F(1,10) = 14,45$, $p < 0.01$) due largely to the sharp reduction in RMS of the unaffected arm when the target can be seen.

Direction of Deviation from Target

The outcome of individual trials witnin a condition were plotted for each subject, but no systematic pattern of drift from target was found. The distribution of mean (x,y) coordinates, for each condition and each subject is presented in Figure 2.2.3. Left hand results were normalised to the right hand and a numerical summary of the quadrant distribution is given in Figure 2.2.4. This snows that mean responses of the normal arm are fairly evenly distributed over the four quadrants. A chi - square test for frequency of mean response in each of the quadrants revealed no significant unevenness in distribution. However the same test applied to the

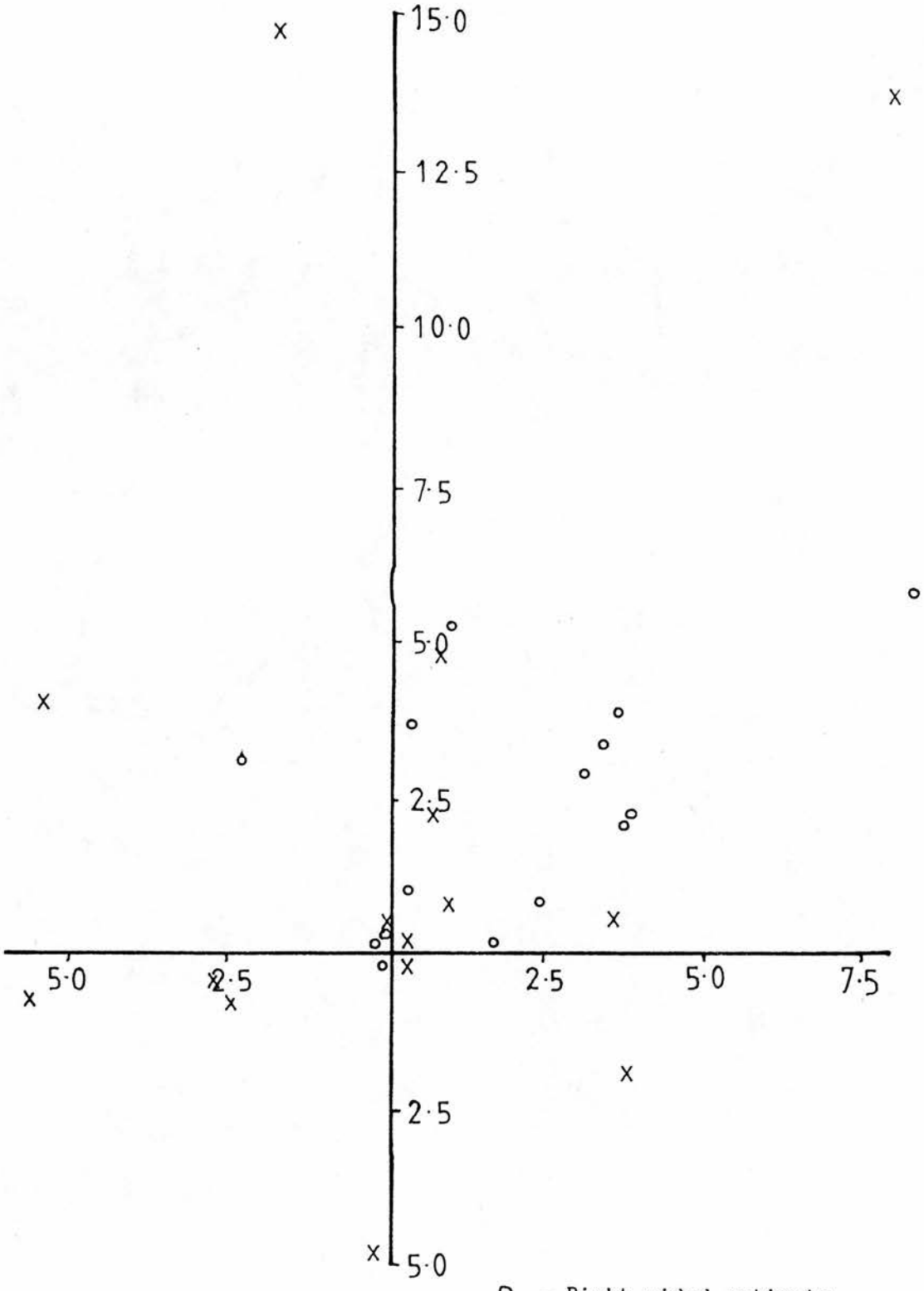
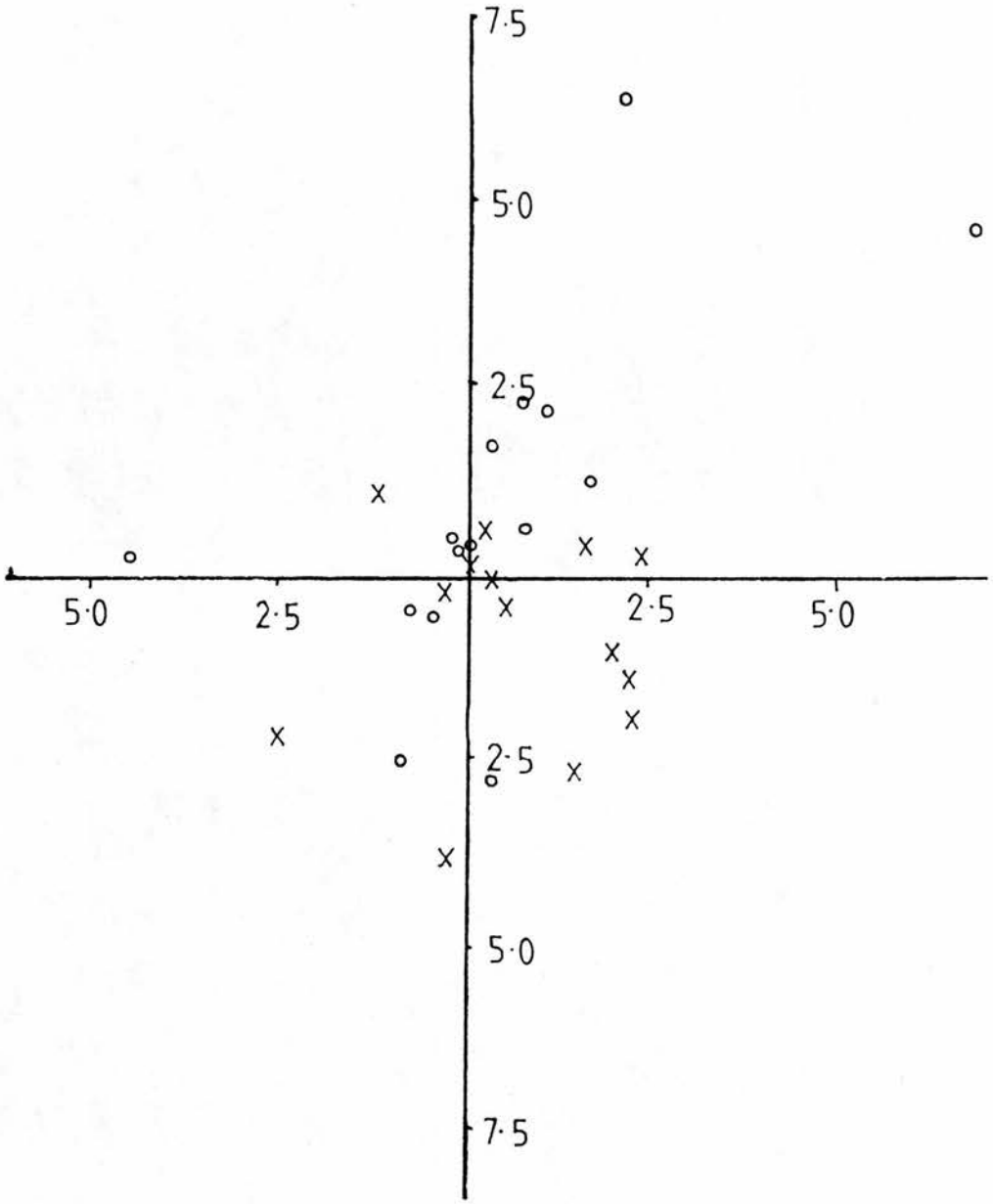



Figure 2.2.3 Distribution of mean points across visual/target conditions of Experiment 2.2 for GA arm condition.




o - Right hand used.
X - Left hand used.

Figure 2.2.3 Distribution of mean points across visual/ target conditions of Experiment 2.2 for NA arm condition.

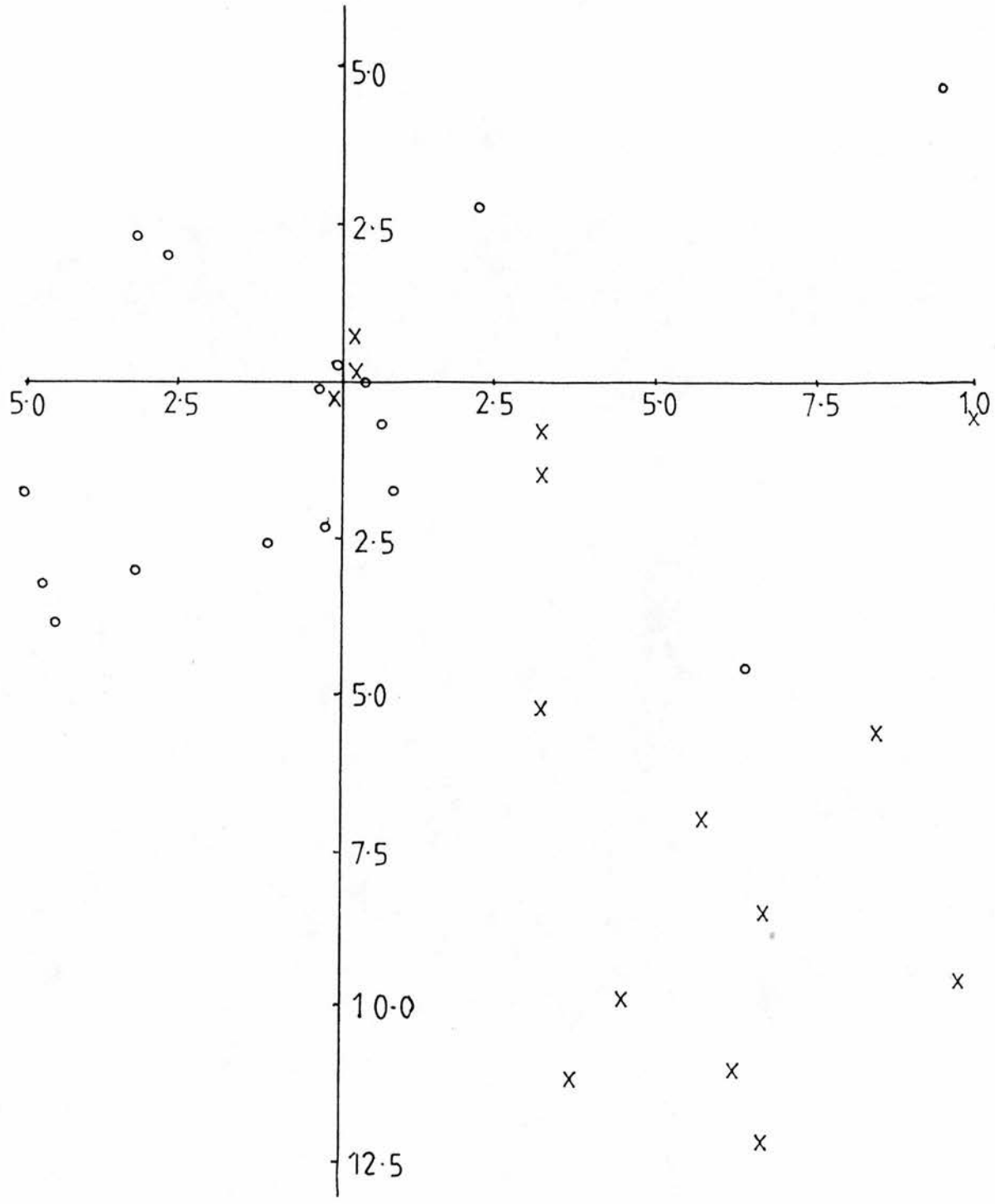
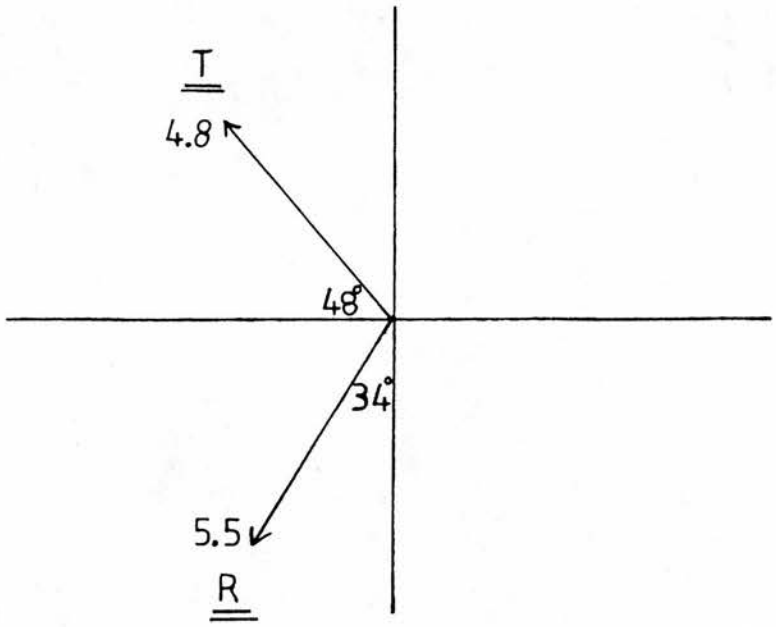



Figure 2.2.3 Distribution of mean points across visual/ target conditions of Experiment 2.2 for AA arm conditions.

| DISTRIBUTION | | ARM |
|---|---|---|
| 7½ | 9 | NA |
| 9½ | 4 | |
| 14 | 9 | GA |
| 4 | 3 | |
| 5 | 2½ | AA |
| 18 | 4½ | |

Figure 2.2.4. Numerical summary of Figures 2.2.3. with left handed points adjusted to right handed.




T : Felt position of the finger of the affected arm when a target for a reach by the unaffected arm.

R : Felt end position of the affected arm when reaching to the cross.

Figure 3.2.5. Vector analysis of the effect of hemiparesis on target position (T) and reaching (R).

unaffected and affected arms showed them to be significantly uneven in distribution at the 5% and 1% levels, respectively. Figure 2.2.4 illustrates affected arm responses are concentrated in the lower left quadrant, whilst unaffected arm responses are found mainly in the upper two quadrants; in particular the upper left one. Following the procedure outlined in Experiment 2.1, vectors were calculated to assess the effects of hemiparesis on the limb wnen moving and when acting as a target, using group means from four of the movement/target conditions:-

1) Unaffected arm to affected finger under NV.

2) Unaffected arm to cross under NV.

3) Affected arm to cross under V.

4) Unaffected arm to cross under V.

The vector expressing the effect of hemiparesis on target position was calculated from (1) and (2), with (2) acting as the origin. The other vector was obtained from (3) and (4), with (4) as the origin. The two vectors are displayed in Figure 2.2.5 and indicate that the effect of hemiparesis on movement is an undershoot of the exteroceptive target. However when the hemiparetic arm is the proprioceptive target, unaffected performance errs in the direction of an overshoot.

## Discussion

The observation in Experiment 2.1, that under conditions permitting full visual monitoring, the hemiparetic arm can localise a target as accurately as the unaffected or normal arm, is confirmed by the results presented here. However across the experimental conditions, both the ffected and unaffected limbs depart from the normal level of performance.

The only difference between the unaffected and normal arm is on pointing to the proprioceptive target when the target cannot be seen. Witn sight of the target, both arms err only slightly and are consistent in response to the two types of target. This demonstrates that when the target, but not the reaching arm, can be seen guidance of reaching is quite accurate. With tne normal arm, substitution of a visually defined target by a somatic, proprioceptively defined target, does not alter perception of target position. However, unaffected arm performance deteriorates markedly and is even slightly worse than when information concerning target location can only come from memory. This demonstrates the unreliability of somatic proprioception in the affected arm. This conclusion is supported by affected arm performance itself. Constantly providing the affected arm with contralateral somatic information about target position leads to an improvement in response

consistency and a marginal increase in accuracy.

Figures 2.2.4 and 2.2.5 demonstrate that when proprioceptive information from the affected arm is attended to as a source for control, it produces a systematic directional effect. As in Experiment 2.1, the affected arm under-extends and over-abducts. The mechanisms underlying this deviation also appear to act upon the felt position of the arm when static, since the directional error produced by the unaffected arm when pointing to the affected finger mirrors this misperception. This implies the affected arm to be felt to be more extended and adducted than it truly is.

## Experiment 2.3 - The effect of varying the amount of visual control on repetitive reaching

### Introduction

The previous experiment demonstrated the necessity of visual monitoring of limb movement for accurate, performance with the affected arm. In this experiment, the question to what extent is this dependent on continuous visual monitoring is examined, by keeping the target constantly visible across conditions which vary in the directness and timing of visual registration of the arm. This is done in two ways. Timing is varied either by allowing the subject sight of his starting position on each trial (SP), or by showing him his error on a trial, prior to commencement of the next trial (KR). The directness of visual monitoring is varied in that in the KR and SP conditions the subject does not actually see his arm. Two further conditions, SP+ and KR+, additionally permit sight of the arm at the start and end of each trial, respectively.

The major question addressed in this experiment is therefore, do patients need to see their affected arm at

all times during the task movements for accurate performance? Or will the provision of end-point error or starting point information be sufficient to correct for errors in performance. From studies on linear positioning tasks (eg Faust-Adams, 1975) it might be expected tnat by giving the patient knowledge of results (KR) about his movements, performance would improve.

## Methods

### Subjects

The same subjects participated as in Experiment 2.2

### Apparatus

The apparatus of the previous experiment was used with slight modification which is described below.

### Design

The design was mixed and of two factors: Arm x Vision. The arm conditions were as in the previous experiment and the exteroceptive target of that experiment was always used. The visual conditions were:-

SP - The position of the pointer was visible at the start of each trial but the arm could not be seen.

SP+ - The arm and body could be seen throughout the block of trials except when the arm was partially occluded by the table top.

KR - Through visual inspection of the marked end point of each trial, subjects were permitted to assess their error.

KR+ - When receiving KR the arm remained visible after responding.

KRSP - A combination of KR and SP in which the arm remained unseen.

### Procedure

The locations for the experiment, time constraints, activity limits, familiarisation and practice routines,

ordering of conditions, requirements made of subjects, number of trials per condition and procedure in general, were the same as in Experiment 2.2.

Information about starting position was given by loosening the nem of the cloak, attached to the table, around the subject's normal starting position. During trials he was instructed to poke the pointer up into sight each time the hand was at that position. The SP+ condition was created by removing the cloak.

The perspex table top, screened by a hinged cardboard "lid" on which was marked the target point, was used in the KR condition. After the subject had completed a trial and tne end position marked on the acetate under-surface, tne screen was raised and the mark indicating end point of that trial was revealed. In the KR+ condition a similar procedure was adopted. In this case, the acetate sheet was attached to the top surface f the table. When the subject finished each trial he was asked to keep his arm in that position, the screen was lifted and the experimenter marked the pointer position. The subject could therefore see his position with respect to the target and also his arm.

## Results

### Scalar Error

Individual means, group means and standard deviations are presented in Table 2.3.1 with the group means and standard deviations plotted in Figure 2.3.1. The results were analysed using three ANOVA's:-

1) A repeated measures Arm x Vision (2 x 6) ANOVA comparing unaffected and affected arms.

2) A mixed ANOVA (2 x 6) Arm x Target ANOVA comparing the unaffected with the normal arm.

3) A one - way repeated measures ANOVA with six levels to examine normal arm performance alone.

A main effect of Arm was found in the first ANOVA ($F(1,5) = 7.33$, $p < 0.05$) indicating the unaffected arm to be the more accurate. There was also a main effect of Vision ($F(5,25) = 7.01$, $p < 0.001$) largely due to the the difference between the FV condition and all other conditions. This is particularly the case for the affected arm, as none of the impoverished visual conditions significantly differ from each other.

Although the Arm x Vision interaction just escapes significance at the 5% level ($F(5,25) = 2.32$, $p = 0.07$) there is a trend in the unaffected arm results for the KR conditions to approach the level of accuracy found under FV. Indeed on post - hoc analysis the KR+ condition does

Table 2.3.1
Mean Scalar Error (cm)

NORMAL SUBJECTS

| CONDITION | N1 | N2 | N3 | N4 | N5 | N6 | GROUP |
|---|---|---|---|---|---|---|---|
| FV | 0.40 | 0.38 | 0.18 | 0.05 | 0.31 | 0.15 | 0.21 (0.11) |
| KR | 0.88 | 0.52 | 0.29 | 0.46 | 1.20 | 0.70 | 1.19 (0.75) |
| SP | 1.29 | 1.51 | 2.50 | 2.61 | 1.50 | 1.76 | 2.22 (1.38) |
| KR+ | 0.21 | 0.32 | 0.56 | 0.50 | 0.52 | 0.21 | 0.59 (0.25) |
| SP+ | 1.75 | 1.36 | 3.96 | 2.52 | 2.68 | 1.85 | 2.27 (0.99) |
| KRSP | 0.57 | 0.50 | 0.75 | 0.53 | 0.36 | 0.57 | 0.79 (0.36) |

HEMIPARETIC SUBJECTS

| CONDITION | H1 | H2 | H3 | H4 | H5 | H6 | GROUP |
|---|---|---|---|---|---|---|---|
| FV | 0.21 | 0.10 | 0.29 | 0.07 | 0.67 | 0.11 | 0.24 (0.23) |
| KR | 1.26 | 2.22 | 0.97 | 8.79 | 5.51 | 1.44 | 3.36 (3.14) |
| SP | 5.73 | 5.74 | 4.69 | 6.19 | 5.65 | 2.08 | 5.01 (1.52) |
| AA | | | | | | | |
| KR+ | 0.75 | 1.17 | 0.83 | 8.23 | 5.94 | 1.61 | 3.09 (3.20) |
| SP+ | 1.65 | 4.75 | 4.40 | 2.79 | 3.58 | 3.00 | 3.36 (1.14) |
| KRSP | 1.10 | 1.24 | 0.64 | 6.76 | 6.03 | 2.65 | 3.07 (2.67) |
| FV | 0.21 | 0.16 | 0.23 | 0.16 | 0.41 | 0.11 | 0.21 (0.11) |
| KR | 1.11 | 1.06 | 0.85 | 1.52 | 2.40 | 0.16 | 1.69 (0.75) |
| SP | 1.82 | 4.92 | 1.86 | 2.77 | 1.26 | 1.20 | 2.22 (1.38) |
| UA | | | | | | | |
| KR+ | 0.77 | 0.60 | 0.51 | 0.37 | 0.98 | 0.32 | 0.59 (0.25) |
| SP+ | 3.12 | 3.79 | 1.30 | 1.96 | 1.75 | 2.20 | 2.27 (0.99) |
| KRSP | 1.14 | 0.85 | 0.89 | 0.29 | 0.96 | 0.29 | 0.74 (0.36) |

CONDITION - For explanation of abbreviations see page 115.

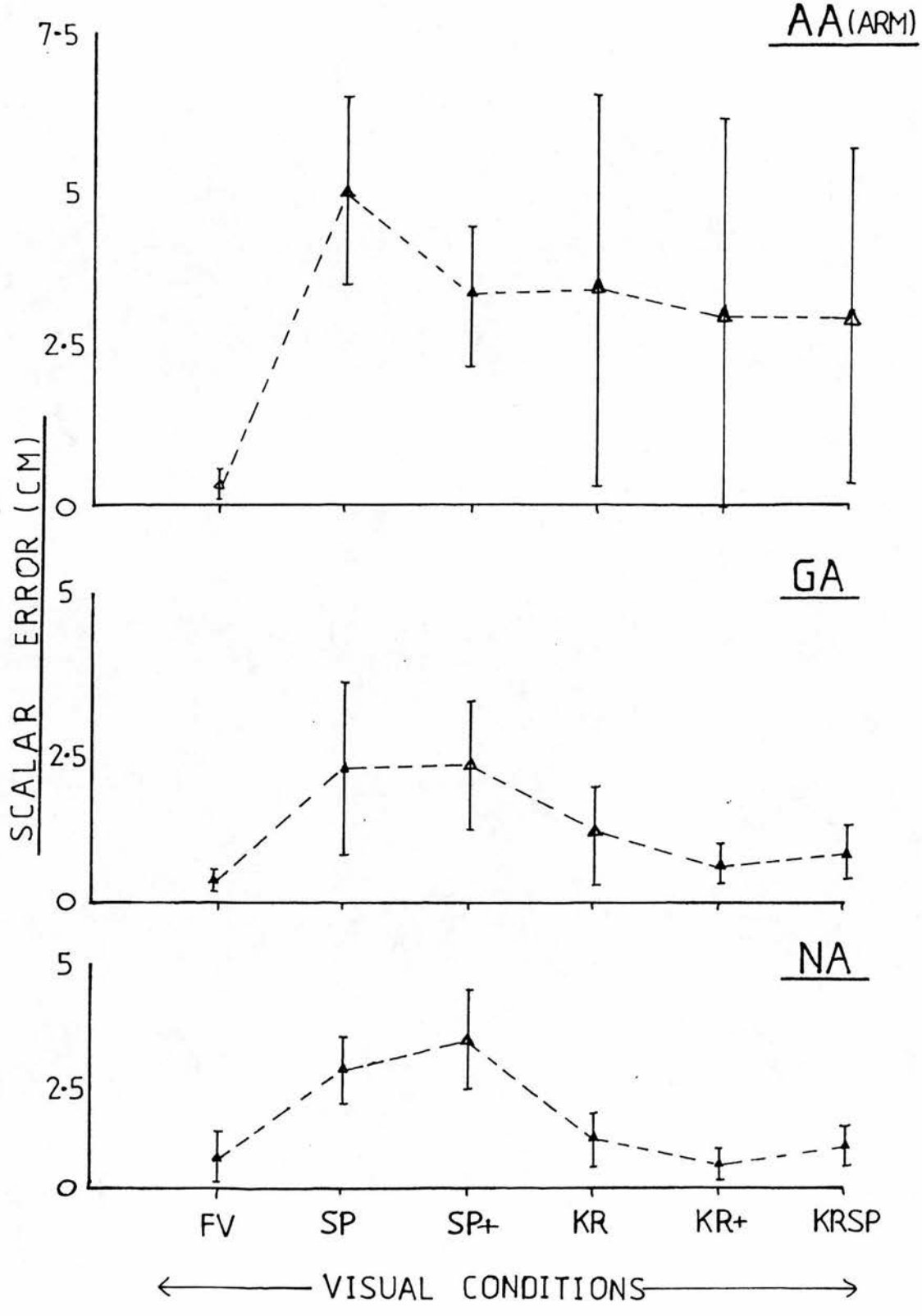



Figure 2.3.1. Average Scalar Error.

Table 2.3.2
RMS Error (cm)

| | | | | NORMAL SUBJECTS | | | |
|---|---|---|---|---|---|---|---|
| CONDITION | N1 | N2 | N3 | N4 | N5 | N6 | GROUP |
| FV | 1.14 | 0.69 | 0.35 | 0.44 | 0.67 | 0.29 | 0.60 (0.32) |
| KR | 1.84 | 1.41 | 1.27 | 2.01 | 1.53 | 1.44 | 1.59 (0.28) |
| SP | 0.89 | 0.84 | 1.17 | 1.02 | 0.92 | 1.30 | 1.02 (0.18) |
| KR+ | 1.04 | 0.68 | 0.85 | 1.14 | 1.26 | 0.81 | 0.98 (0.21) |
| SP+ | 0.95 | 1.11 | 1.18 | 1.21 | 0.99 | 1.04 | 1.08 (0.11) |
| KRSP | 1.07 | 1.31 | 1.03 | 1.21 | 1.65 | 1.06 | 1.22 (0.25) |

| | | | | HEMIPARETIC SUBJECTS | | | |
|---|---|---|---|---|---|---|---|
| CONDITION | H1 | H2 | H3 | H4 | H5 | H6 | GROUP |
| FV | 0.50 | 0.42 | 0.44 | 0.69 | 0.96 | 0.27 | 0.58 (0.28) |
| KR | 2.10 | 2.16 | 1.84 | 3.91 | 2.81 | 1.93 | 2.46 (0.79) |
| SP | 1.86 | 1.52 | 1.36 | 2.65 | 2.83 | 1.44 | 1.86 (0.69) |
| AA KR+ | 1.50 | 1.16 | 1.57 | 3.96 | 2.44 | 1.73 | 2.06 (1.03) |
| SP+ | 1.28 | 1.11 | 0.99 | 2.34 | 2.66 | 1.14 | 1.59 (0.73) |
| KRSP | 1.77 | 1.24 | 1.78 | 3.08 | 2.97 | 2.02 | 2.15 (0.73) |
| FV | 0.38 | 0.37 | 0.37 | 0.80 | 0.74 | 0.24 | 0.48 (0.23) |
| KR | 1.52 | 1.87 | 1.15 | 2.25 | 1.95 | 1.21 | 1.51 (0.40) |
| SP | 1.06 | 1.36 | 1.00 | 1.54 | 1.63 | 0.87 | 1.25 (0.31) |
| UA KR+ | 1.07 | 0.73 | 1.05 | 2.43 | 1.71 | 1.31 | 1.38 (0.61) |
| SP+ | 1.12 | 1.13 | 1.06 | 1.33 | 1.55 | 1.22 | 1.24 (0.18) |
| KRSP | 0.95 | 0.88 | 1.05 | 2.46 | 1.88 | 1.42 | 1.44 (0.62) |

CONDITION - for explanation of abbreviations see page 115.

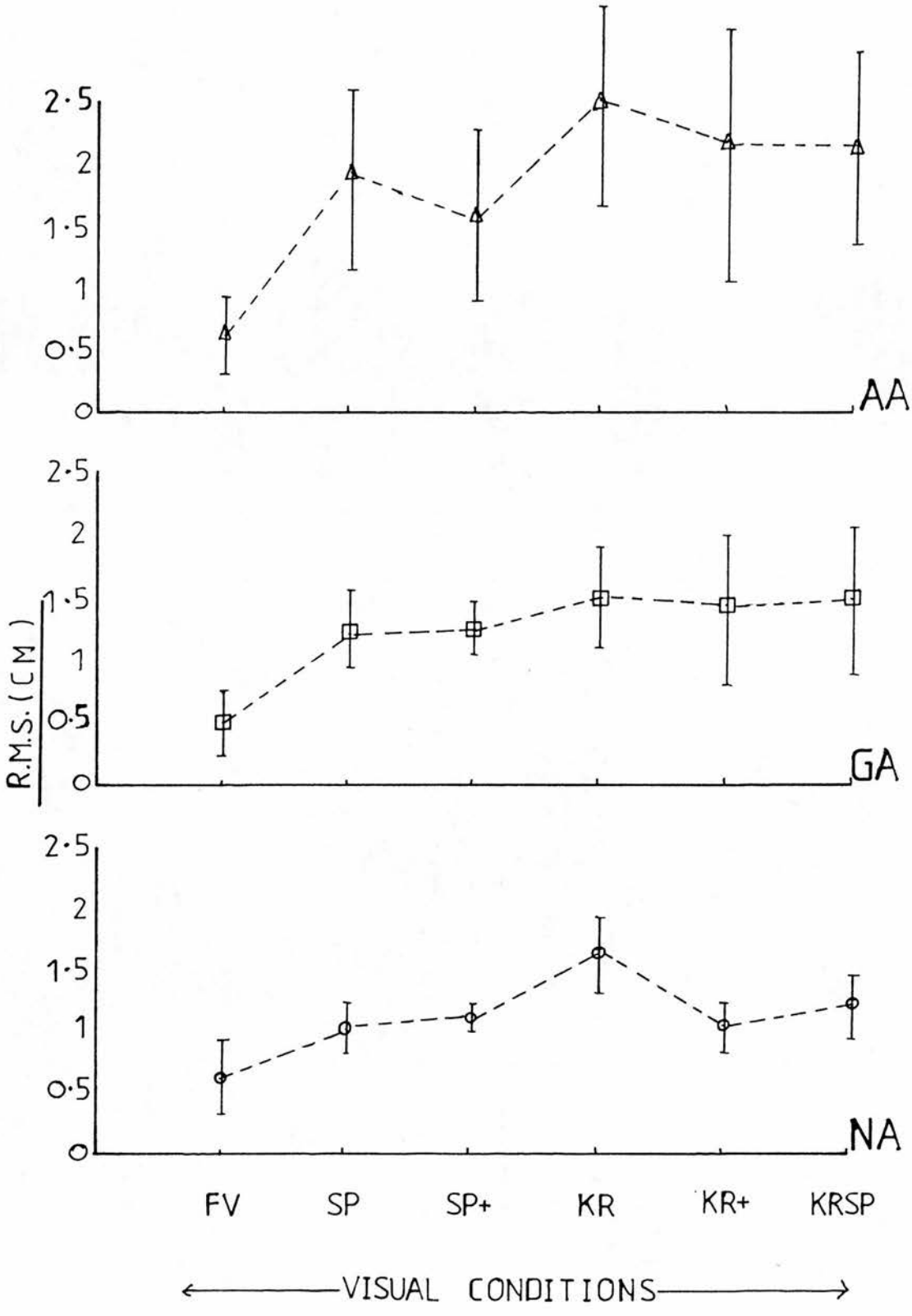



Figure 2.3.2. Mean R.M.S.

not significantly differ from the FV condition, whilst both the - conditions do. However, there is also no difference between the SP and KR results.

A similar pattern of results is evident for the normal arm. On ANOVA (2), there is no main effect of Arm, but there is a main effect of Vision ($F(5,5) = 25.23$, $p < 0.001$) which is also present in ANOVA (3) ($F(5,25) = 26.00$, $p < 0.001$). Post-hoc analysis reveals a clear separation between the SP and KR conditions with no significant difference between the latter and the FV condition.

Root Mean Square Error

Individual means, group means and standard deviations are presented in Table 2.3.2 with the group data displayed in Figure 2.3.2. Analysis of the results was conducted using the ANOVA's outlined on page x.

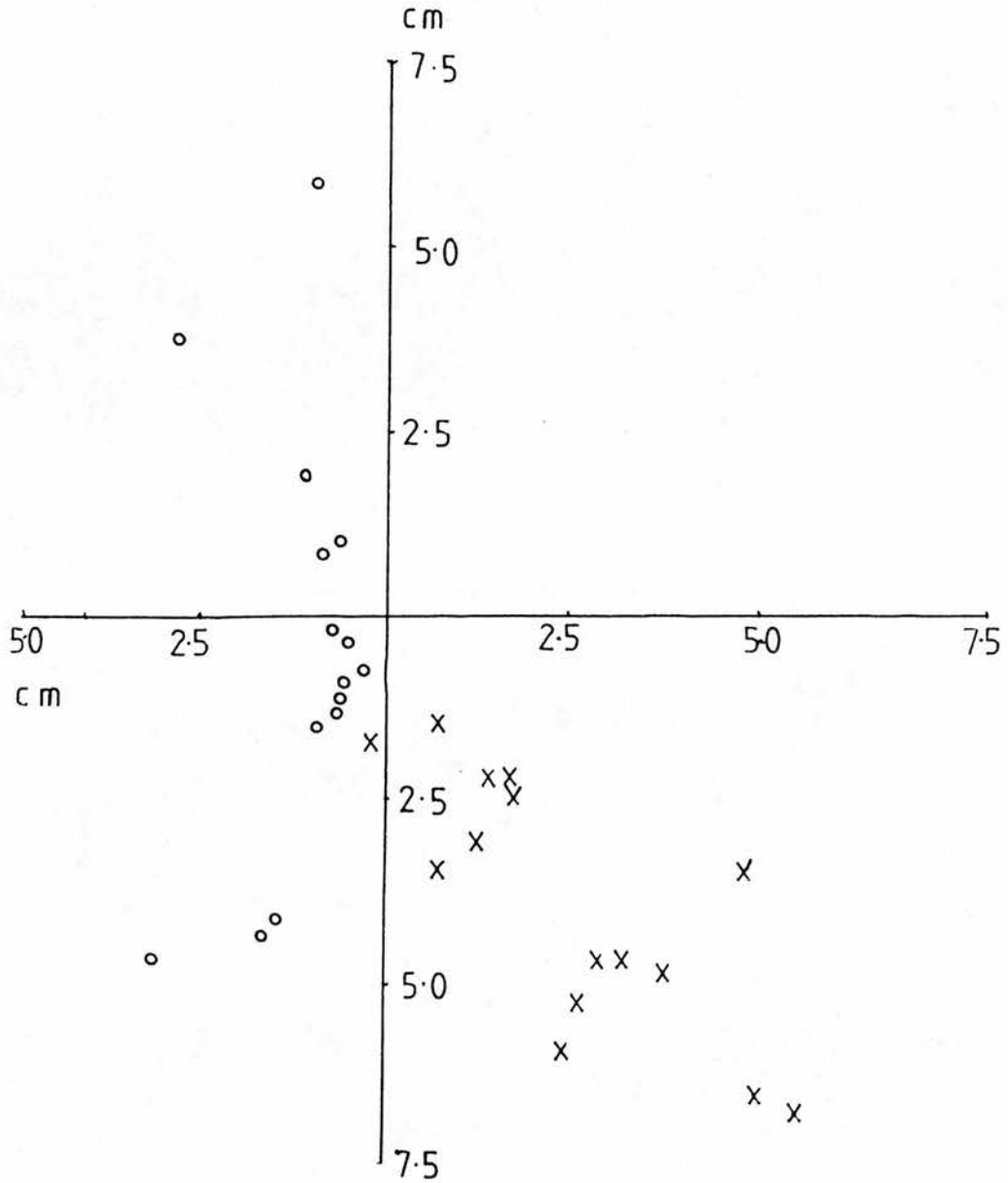
On ANOVA (1), there is a main effect of Arm ($F(1,5) = 18.37$, $p < 0.01$) and of Vision ($F(5,25) = 19.67$, $p < 0.001$) with a significant interaction between these two factors ($F(5,25) = 8.05$, $p < 0.001$). As Figure 2.3.2 shows, these outcomes are due to the impoverished visual conditions producing less consistent performance, more so for the affected arm.

The only significant result from ANOVA (2) is a main

effect of Vision ($F(5,5) = 21.56$, $p < 0.001$) indicating the unaffected and normal arms are comparable in consistency of performance, with both becoming less so when sight of the arm is manipulated. This point is reinforced for the normal arm in ANOVA (3) by a main effect of Vision ($F(5,25 = 11.63$, $p < 0.001$).

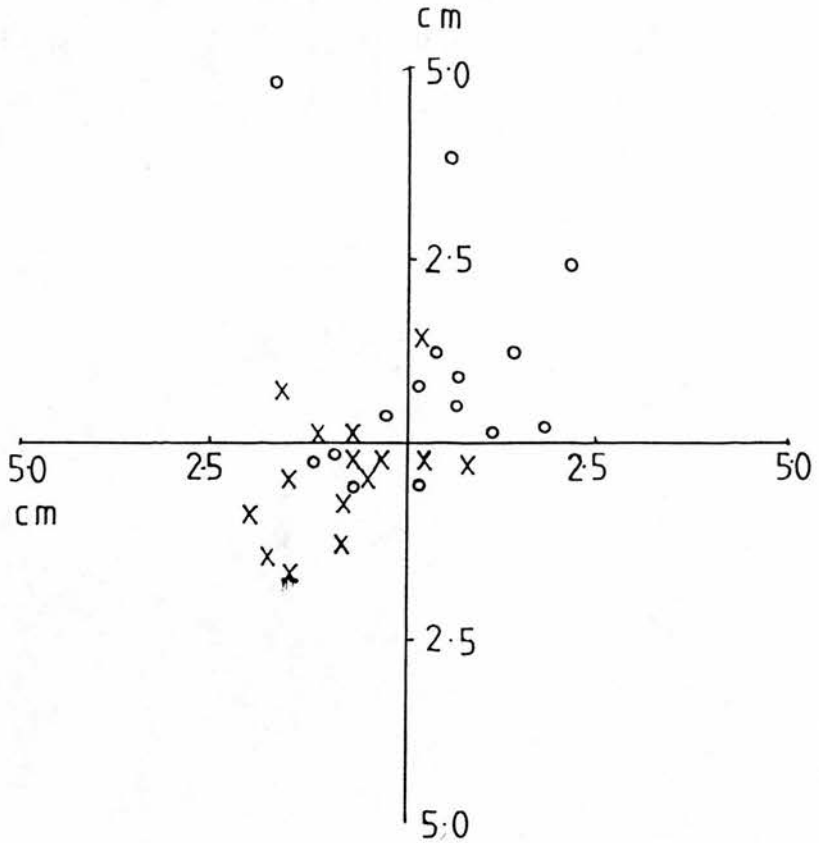
Direction of Deviation from Target

As in the previous two experiments a consistent pattern of drift across trials was sought, but not found. The distribution of mean (x,y) coordinates for each subject in each condition is plotted in Figure 2.3.3. A summary of quadrant distribution is presented in Figure 2.3.4 with left hand results normalised to right. Chi - square tests applied to these distributions indicate a significant unevenness of distribution only for the affected arm and, as is evident from the Figure, this is due to the concentration of affected arm responses in the lower left hand quadrant. Following the procedure outlined in Experiment 2.2, vectors were calculated from the mean group results to illustrate the effect of hemiparesis on reaching. This was done using the difference between the unaffected and affected arm coordinates, with the unaffected result as the origin. The five vectors are displayed in Figure 2.3.5. All show a deviation into the lower left hand quadrant. However, the horizontal component of this is more marked in the KR conditions and in the SP+ condition the vector is almost vertical.



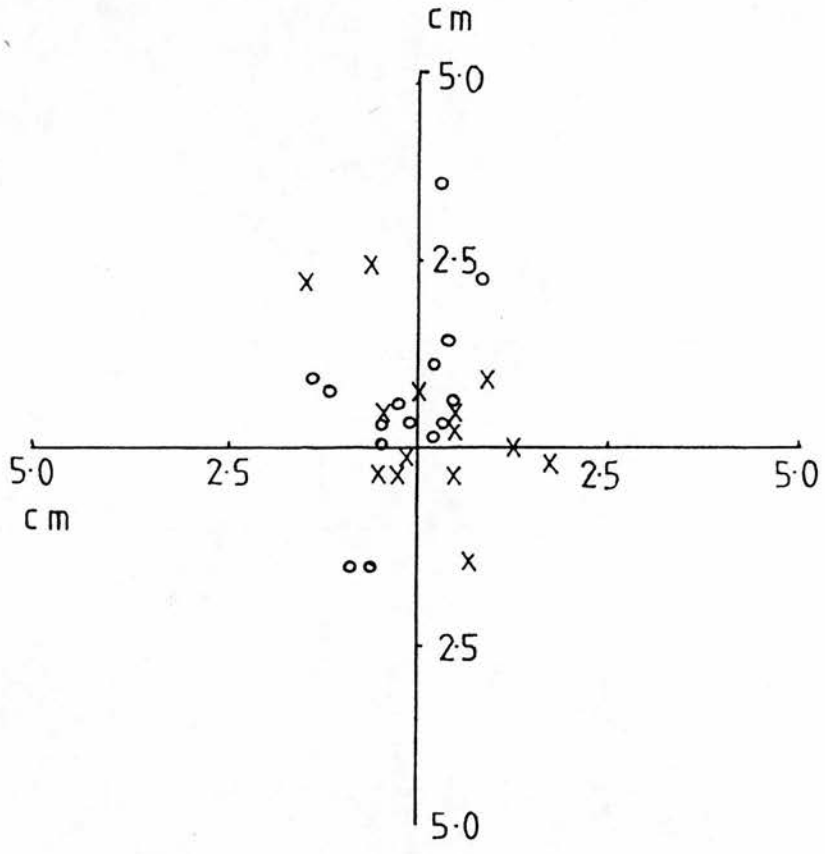

O - Right hand used.
X - Left hand used.

Figure 2.3.3 Distribution of mean points across visual conditions of Experiment 2.3 for AA arm condition.




O - Right hand used.
X - Left hand used.

Figure 2.3.3. Distribution of mean points across visual conditions of Experiment 2.3 for GA arm conditions.




O - Right hand used.
X - Left hand used.

Figure 2.3.3. Distribution of mean points across visual conditions of Experiment 2.3 for NA arm conditions.

| DISTRIBUTION | | ARM |
|---|---|---|
| 9½ | 10½ | NA |
| 7 | 3 | |
| 12 | 3 | GA |
| 10 | 5 | |
| 5 | 0 | AA |
| 24 | 1 | |

Figure 2.?.4. Numerical summary of Figures 2.3.3. with left handed points adjusted to right handed.

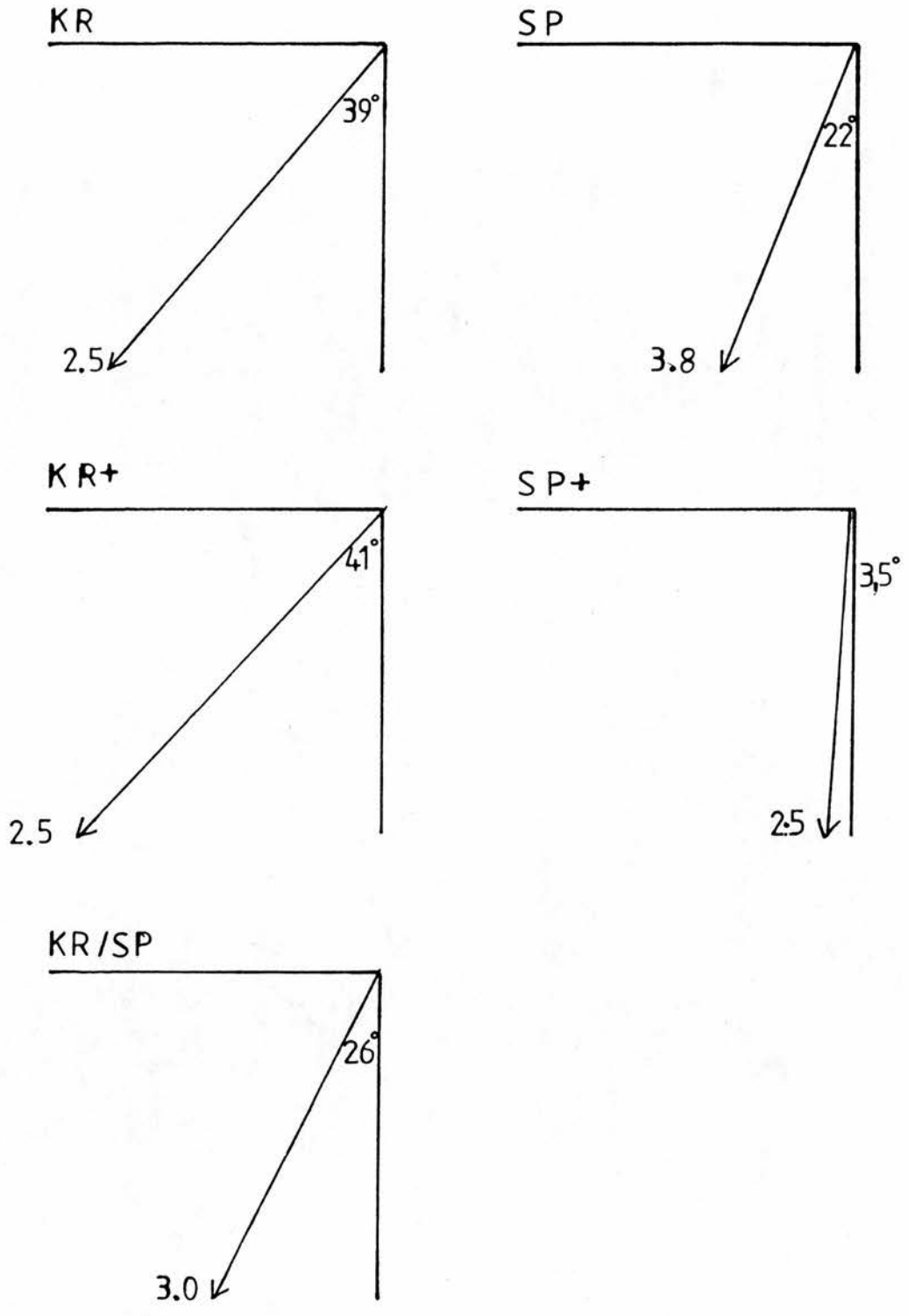



Figure 2.3.5. Vector analysis of the effect of hemiparesis on reaching.

Discussion

When KR is provided after each trial the normal and unaffected arms are as accurate, but less consistent, than when full visual monitoring is permitted. The underlying control processes for these limbs can therefore make use of retrospective error information in the production of movement. Figures 2.3.4 and 2.2.5 confirm the results of the other two experiments, in showing that the paretic limb errs in the direction of under-extension and over-adduction. There is little evidence that KR can reduce the extent of this deviation using visual feedback of end position. This then indicates terminal feedback is not sufficient, continuous visual monitoring may be essential. However when the individual means are examined (Table 2.3.1) it becomes apparent that the high group mean is largely due to the poor performance of the two of the right hemisphere lesioned subjects. Therefore this conclusion must be treated with caution. More will be discussed concerning the performance of the right hemisphere lesioned patients in the concluding section of this chapter.

Simply allowing the subject to see the hand before the start of each trial, does not produce an improvement in accuracy. However there is some evidence from the SP+ condition that the patients may have visually monitored

arm position with respect to the table, to correct for overadduction. This would explain the reduced laterality in that vector and the slight, although non-significant, reduction in accuracy.

General Discussion

The principal result across all the experiments reported in this Chapter is the effect of stroke on the subject's perception of the position of the affected arm. In the absence of vision, kinesthetic information misleads the system resulting in a regular pattern of misperception. The affected arm is felt to be more extended and less abducted than it really is. That this represents a shift in somatic proprioception, is evident from the direction of error observed when the unaffected arm reaches to the index finger of the affected side in Experiment 2.2. What mechanism could be underlying this effect?

Goodwin et al (1972) demonstrated in normal subjects, that wnen the biceps tendon was vibrated the forearm was perceived to be more extended than it actually was. Capaday and Cooke (1981) further investigated this phenomenon by questioning whether muscle vibration during movement could affect the course of movement and the intended final limb position. They examined flexion and extension of the elbow in a step tracking task, with and without sight of performance and target attainment.

Without visual guidance, vibration of the triceps applied either continuously over trials or only during

individual movements, resulted in undershoot of the flexion target, but had no effect on attainment of the extension target. Similarly when the biceps was vibrated, extension was undershot, but flexion remained unaffected. Therefore Capaday and Cooke (1981) demonstrated that vibration of the muscle antagonistic to the performed movement resulted in an undershoot of the intended target position, whereas vibration of the prime mover had no effect. Under visual guidance these effects were overcome.

Together, the studies of Goodwin et al (1972) and Capaday and Cooke (1981), demonstrate a vibration induced systematic distortion of felt arm position, which is similar in behavioural outcome to the results presented in this Chapter. Burke et al (1976) have demonstrated that vibration activates the muscle spindles of the vibrated muscle. On the basis of this result, Capaday and Cooke (1981) suggest that afferent activity from the spindles of the antagonist is monitored by the CNS during movement. As support for this postulate they cite the work of Hagbarth et al (1975). During movement the antagonist is stretched. Hagbarth et al (1975) demonstrated that during the course of its lengthening, the spindles of the antagonist become active but spindle activity in the agonist is silenced.

Capaday and Cooke's postulate fits well with

Bobath's (1978) developing spasticity hypothesis. In spastic muscles it has been demonstrated that the muscle spindles are hyperexcitable (Ashby and Verrier,1976). Therefore at rest and during movement, it is likely that the Ia afferents are over-active or over effective centrally. If during extension the biceps has, or is developing spasticity, then there will be an abnormally high amount of antagonist proprioceptive information which, as Capaday and Cooke argue, could misinform the CNS about the degree of elbow extension in a manner in agreement with the results presented here.

The experiments in this Cnapter suggest one of the functions of vision in the control of movement following stroke, is to counter-act any misinformation provided by, or misperception of, somatic proprioception. In doing this, Experiment 2.3 demonstrated that continuous visual guidance throughout performance appears necessary. Also, the vector analysis of the SP+ condition in that experiment appears to demonstrate improved control of the shoulder adduction component of the reach when the subject can see the part of the arm unoccluded by the table. Therefore consciously or otherwise, the processes controlling the affected arm can utilise information picked up visually. This concerns the angle of the humerus to both the edge of the table and the body with respect to the relationship of the body midline to the target, to prevent over-adduction at the shoulder. The

issue of continuous visual guidance and its potential for improved control of the affected arm is further explored in the next Chapter.

Before closing this Chapter, a word needs to be said about the experimental design employed. It has become a tradition in the motor skills literature to use the differences between group means to infer the effects of experimental manipulations on motor responses. Such an approach was adopted in this Chapter. However typically, studies of normal skill utilise much larger sample sizes, the idea being that a large n will minimise inter-individual variability. It was practically impossible to run a large sample of patients and as the standard deviations about the group means demonstrate, there was a high degree of inter-patient variability. The pitfalls of this approach to meaningful assessment of performance was indicated in Experiment 2.3. In that experiment, the poor results of two of the subjects could have elevated the group mean data to such an extent, that the improvement in performance under K.R. conditions by the other four subjects was not statistically significant. In the following Chapter an attempt is made to take individual differences into account in interpreting performance.

On this point it is interesting to note that all the right hemisphere lesioned patients had poorer shoulder

movement than the left hemisphere lesioned patients, (Table 2.1.1 and page 89). They also tended to make larger errors than the left hemisphere lesioned patients, (Tables 2.1.2 and 2.1.3). In a review of work on hemispheric specialisation Trevarthen (1981) presents evidence which indicates that the right hemisphere has a more bilateral representation of body image, and is more concerned with spatial coordination than the left hemisphere. A defect in these functions could explain the poorer shoulder control and higher error scores exhibited by the right hemisphere lesioned subjects.

# Chapter III

## Linear Positioning Tasks

## Introduction

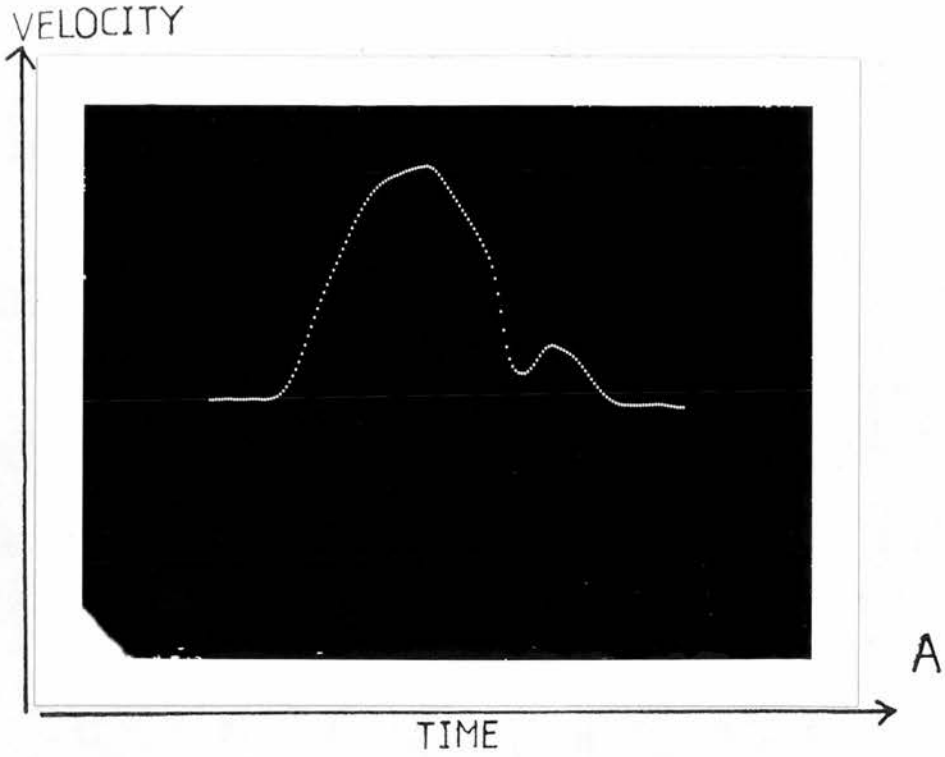
In the previous Chapter, the performance of the upper limb following stroke was assessed solely in terms of end-point accuracy of movement. Whilst this approach has proved extremely fruitful in furthering our understanding about motor control, it should be recognised that it has its limitations. Simply recording the end result of a movement tells us little about the motor processes involved in the execution of the act which gives this result. For example, from the results of the full vision condition of Experiments 2.2 and 2.3, hemiparetic performance would appear normal. However when observing the patients performing the task, it was noticeable that their movements in reaching to the target were clearly abnormal. Whilst the unaffected and normal limbs appeared to move quickly and smoothly to the target, the hemiparetic action was slow and halting. End-point accuracy alone cannot comment on this aspect of performance. Another example of its limitations comes from consideration of the results in the no-vision conditions. Although evidence was provided concerning the degree and consistency of the affected arm undershooting the target, there is no data on how such errors were made. Therefore in order to study the quality of movement and gain further insight into the dysfunction in control caused by stroke, a kinematic

analysis is necessary.

The tasks employed in this Chapter are variants of the linear positioning task (discussed on page 40). Whereas in the traditional linear positioning task, a criterion and then reproduction movement are executed, the experiments in this Chapter are restricted to the performance of so-called criterion movements. This is because the objective is to elucidate the processes underlying motor performance not motor memory and also because, as Newell (1976) points out, the typical linear positioning task halves the decision processes required of the subject.

The experimental set - up, which is fully described on page 152, consisted of a track along which subjects had to move and position a wrist cradle. It was modelled on a construction used by Wadman (1979) and designed to record movements which are largely uni-axial involving the elbow. Built into this equipment were sensors which could pick up both the position and velocity of the cradle. Therefore for any given trial, information was available not only on any end-point error made by the subject, but also, from the velocity profile, on the nature of the movement trajectory leading up to the end point.

The examination of movement trajectories is



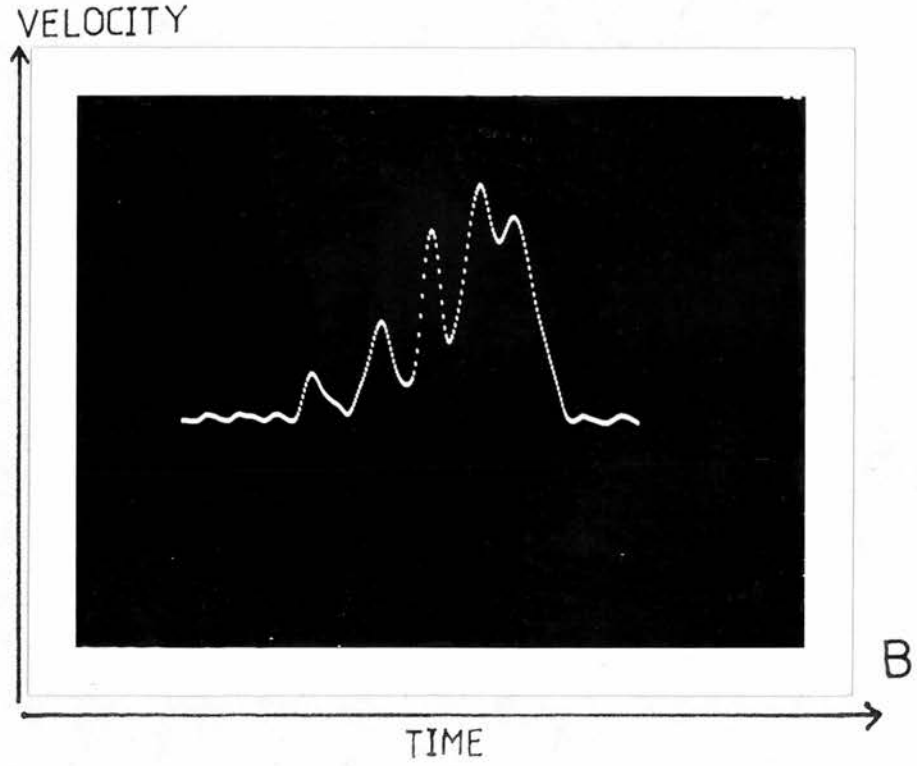




Figure 3.1 - Showing velocity profiles obtained from the tachogenerator in Experiment 3.1

A : Extension of the normal arm

B : Extension of the affected arm (Subject WI).

increasingly proving itself a valuable tool in the study of motor control. The method employed in this Chapter is derived from Brooks (1974) and consists of partitioning the trajectory into its component sub-movements on the basis of the number of peaks present in the velocity profile as illustrated in Figure 3.1. Following from Brooks (1974), this manner of analysis has been used to assess the degree to which movements are under either preprogrammed or continuous, corrective, control in populations ranging from young infants (von Hofsten, 1980) to normal adults (Jagacinski et al, 1980).

In normal adults, the velocity profile of simple aiming movements typically contains only one large peak, often accompanied by an additional much smaller one at the end of the trajectory. In these movements it has been found that most of the action is planned in advance with the majority of the trajectory, around 93% (Vince, 1948), executed in a preprogrammed, ballistic fashion. This corresponds to the large peak in profile A. The small peak at the finish has been shown to function as a fine correction, usually visually based, serving to precisely home in the effector, once it is in the general "ballpark" (Greene, 1972) of the target region (Carlton, 1981). From the conclusions of Chapter II and the observation that patients' actions are typically jerky, it might be expected that a feature of hemiparesis is a breakdown in the normal trajectory pattern resulting in

actions consisting of multiple sub-movements under continuous control (Profile B).

The velocity profile can also yield precise information on the temporal aspects of the movement. Two of the most studied measures in motor skills research are initiation time (IT) and movement time (MT). IT, in its simplest form, is the time taken from a command to move, to the actual start of movement and therefore reflects the nervous system's speed of translating an intention or plan for action into prescribed movement. MT is the time from commencement to cessation of movement providing information on speed of execution. Consequently it can be used as an indication of the system's ability to produce and sustain the necessary force to transport a limb from a starting position to a goal. The importance of distinguishing between these two time periods is that it allows investigation into whether motor dysfunction following stroke is primarily in the domain of preparing or executing movement.

To complement the study of movement structure, it was decided to conduct electromyographic analysis of agonist/antagonist activity. A principal problem here was how to quantify and thus eventually statistically analyse the EMGs. Since Wachholder's pioneering work (Wachholder and Altenburger, 1926) on the effects of speed demands upon EMG waveforms, two patterns of

agonist/antagonist activity, corresponding to whether a movement is slow or fast, have been repeatedly confirmed (Wallace,1981). In fast movements a typical tri-phasic pattern of agonist on/off, antagonist on/off and agonist on/off, is found. With slow movements the picture is quite different: there is a slow rise in agonist activity with negligible antagonist activity. In the fast movement the three phases are thought to sequentially function to firstly accelerate the limb from rest, then commence deceleration with the final third phase acting to stabilise the limb at the target, (Angel, 1974; Hallett et al, 1975). In slower movements, Marsden et al (1983) have demonstrated that antagonist participation is unnecessary as the visco-elastic properties of the muscle alone, can serve to decelerate movement. What then might be the expected pattern of activity in the hemiparetic arm given the prediction that the movement will be slow and jerky?

If indeed the movements are slower than normal, then it might be expected that they follow the pattern described for slow movements. However if they additionally turn out to be jerky, then there may be a modification to this pattern. The antagonist could remain inactive throughout the course of the movement with the agonist firing in isolated bursts. This scenario corresponds to the hypothesis that following stroke the major problem is a lack of sustained drive to

the alpha motoneurons responsible for agonist activation. Discontinuities would therefore result at the behavioural level due to discrete pockets of firing. On the other hand, agonist activity could be accompanied by a rise in antagonist activity, following the hypothesis that a failure in reciprocal inhibition of stretch reflex induced antagonist activity could be primarily responsible for decelerative interruptions to the smooth flow of movement.

In attempting to distinguish between these two hypotheses there is a problem. In deciding the timing of muscle onset and offset in fast movements, the sharp rise and fall of traces can be easily identified and indeed, in many studies visual inspection of the EMG record serves as the sole method for establishing criteria as to if and when a muscle is active. With slow movements, where there is a slow rise in activity, it is not easy to determine at which point on the rectified slope one can reliably say a muscle is actually active. Pilot work for this Chapter confirmed this to be a real problem and a review of the literature revealed no established solution as to how it might be resolved. Therefore a statistical criterion for activity was adopted.

In typical behavioural experiments, if the score on any individual trial lies outwith two standard deviations of the mean result over all trials, then that result is

considered atypical, due to the intervention of uncontrolled variables and is subsequently rejected from further analysis. A similar procedure was applied here to the analysis of EMG records. The mean and standard deviation was obtained for the resting level of the muscle at the proposed starting point for a set of movements. In analysing the traces resulting from these movements, the muscle was considered active only for the period(s) during which its level of activity exceeded two standard deviations from the mean resting level.

A final word on general procedure needs to be said before presenting the first experiment. In Chapter II, the pitfalls of the traditional motor skills approach of comparing mean scores of groups of subjects when studying movement pathology was discussed. The main argument against this method is the lack of homogeneity in the sample studied and thus the questionable relevance of average results to the performance of any individual member of a heterogeneous population (Hersen and Barlow, 1976; Shallice, 1979). The experiments contained in this Chapter were conducted on a low number of hemiparetic subjects (typically four) with only one normal control. However in contrast to Chapter II, analysis is at the level of the individual subject with discussion centred around consistent similarities or differences amongst the individuals, across the experimental conditions. Whilst this is premised as a more appropriate method for

assessing perceptuo-motor function following stroke there is an additional practical limitation on the number of subjects that can be studied using kinematic techniques. The problem is that this mode of study has the potential for generating vast amounts of data, the reduction and analysis of which is extremely time consuming. Thus one often finds that those concerned with the kinematics of movement are forced for this reason alone to restrict themselves either to complex experiments with low numbers of subjects or on the basis of prior group work at a simpler level, to simple comparisons with a larger sample.

## Experiment 3.1 - The kinematics of visuo-motor control

### Introduction

The principal aim of this experiment was to extend the findings of Chapter II concerning the role of vision in improving accuracy of hemiparetic arm action, by examining the kinematics of the movement and the accompanying agonist/antagonist muscle activity. The main focus of study is therefore on the effect of the presence or absence of visual information upon the spatio-temporal quality of movement production and the relationship between arm and target. As in Chapter II, this task is tackled in a comparative context, with the unaffected limb and the dominant arm of a normal subject also participating in the experimental conditions.

In addition to manipulating vision, the direction and distance of movement was also varied. Extension is compared with flexion because a dominant theme in the stroke rehabilitation literature is that the flexor synergy of the arm recovers prior to the extensor (Twitchell,1951). However there is a major split between two of the most widely used therapeutic regimes on the significance of this prevalence. Brunnstrom (1970) identifies the return of flexion as a definitive stage in the recovery of function and prescribes a series of

exercises to encourage and develop it. She views the re-establishment of normal controlled flexion as a precursor to the return of movement in the extensor synergy. Bobath (1978), on the other hand, greets the return of flexion with much less optimism. Whilst acknowledging that it marks the end of the flaccid period of recovery, she regards its prevalence over extension as pathological and indicative of the major problem faced in stroke rehabilitation: "the release of abnormal relexes". Her programme therefore strives to combat the predominance of flexion through avoiding stimulation of the flexor synergy by immediately trying to modulate and control it whilst simultaneously attempting to elicit extension movements.

This experiment does not aspire to any resolution of this fundamental split between the two doctrines. However as virtually no experimental work has been conducted on this issue the results may provide an interesting preliminary comment. The main purpose of including direction as a factor in the design, is to examine the possibilty that there may be differences between extension and flexion in movement ability and, if this proves to be the case, to assess the implication in terms of control processes governing arm movements in general.

In addition, recording the accuracy of flexion when

vision is withdrawn affords a further opportunity to examine the idea that there may be a miscalibration of somatic proprioception in hemiparesis. If in extension the limb is "felt"to be more extended than it actually is, then in flexion it may be perceived as less flexed than it is, resulting in an overshoot of the target.

## Method

### Design

The subject's task was to position a pointer, mounted above a wrist cradle, under visually specified target positions. A (2x2x2x2) design was employed with each subject taking part in each condition. The four factors were:-

1) Arm - Affected (AA), Unaffected (UA)

2) Direction - Flexion (F), Extension (E)

3) Vision - Full Vision (V), Vision of target only (NV)

4) Distance - Long (L), Short (S)

This yielded a total of sixteen conditions with ten trials per condition. The combination of the first three factors was randomly ordered for each subject. For practical purposes, the distance variable was not included in this ordering, but was randomly presented within any particular condition arising from the combination of the other three factors.

### Subjects

Four hemiparetic subjects participated. All were in-patients at Astley Ainslie Hospital, Edinburgh and all had been diagnosed as having suffered a C.V.A.. Individual particulars are detailed in Table 3.1.1. Only patients without perceptual or comprehensional deficits and without any other motor disability were included. An attempt was made to concentrate selection on those with

Table 3.1.1

| SUB | SEX | AGE | POST-CVA | LESION | SP | SH | ELB | WR | HAND |
|---|---|---|---|---|---|---|---|---|---|
| JG | M | 72 | 7wks | LEFT | Y | 3+ | 3+ | 3+ | 3+ |
| EW | F | 69 | 5wks | RIGHT | Y | 4 | 4 | 4 | 3 |
| MS | F | 55 | 3wks | LEFT | Y | 2 | 2 | 1 | 1 |
| WI | M | 68 | 6wks | LEFT | Y | 4 | 2 | 2 | 1 |

Columns 7-10 are MRC muscle power scalings referring only to those muscle groups necessary for arm extension. The sixth column represents a simple indication of whether (Y) or not (N) spasticity was assessed as interfering with extension.

difficulty in executing elbow movements. Staff in the hospital's Physiotherapy Department were primed with these specifications and notified the experimenter when they came across a suitable patient. At the first meeting the patient's unaffected arm was tested under the experimental procedure in order to assess his ability to understand the task and the accompanying instructions. Having met these criteria, the affected arm was then tested to ascertain whether he could cover the maximum distance required by the task. Normative data was provided by a sixty-four year old woman (AL) with no history of perceptual or motor disorder.

Apparatus

In Figure 3.1.1, a photograph gives a general view of the experimental set-up and this is expressed diagrammatically in Figure 3.1.2. The track consisted of two stainless steel rods mounted in a steel frame which was bolted to a piece of wood. The section of wood was attached by hinges to another section of wood clamped on to a table, thus enabling the track to be rotated through ninety degrees.

Mounted around the track was a wooden frame. The upper surface of the frame consisted of a sheet of perspex, upon which were fixed three strips of cardboard 5mm in width. These served to define; a start position, the short distance target (10 cm), and the long distance

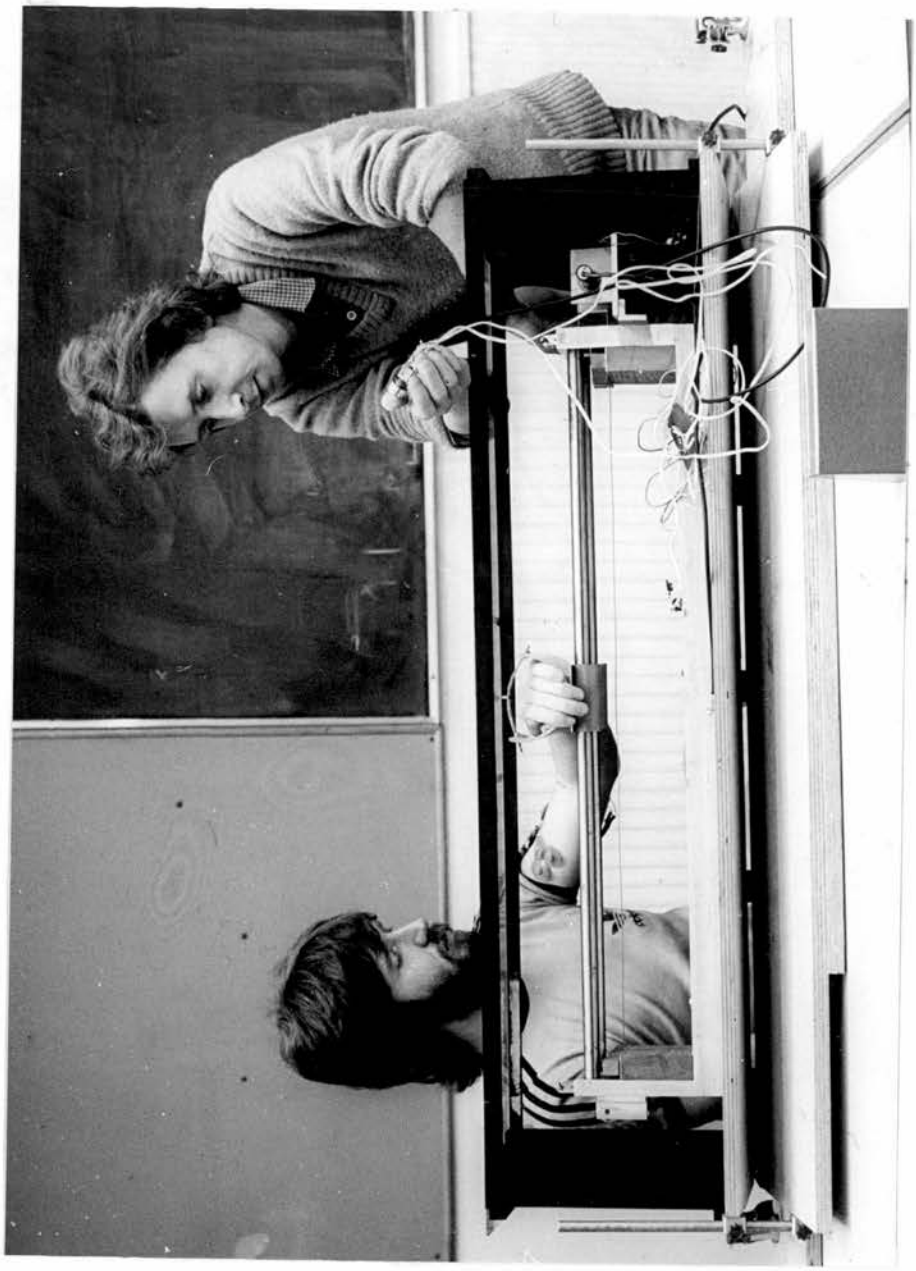


Figure 3.1.1. - The experimental set-up with vision permitted.

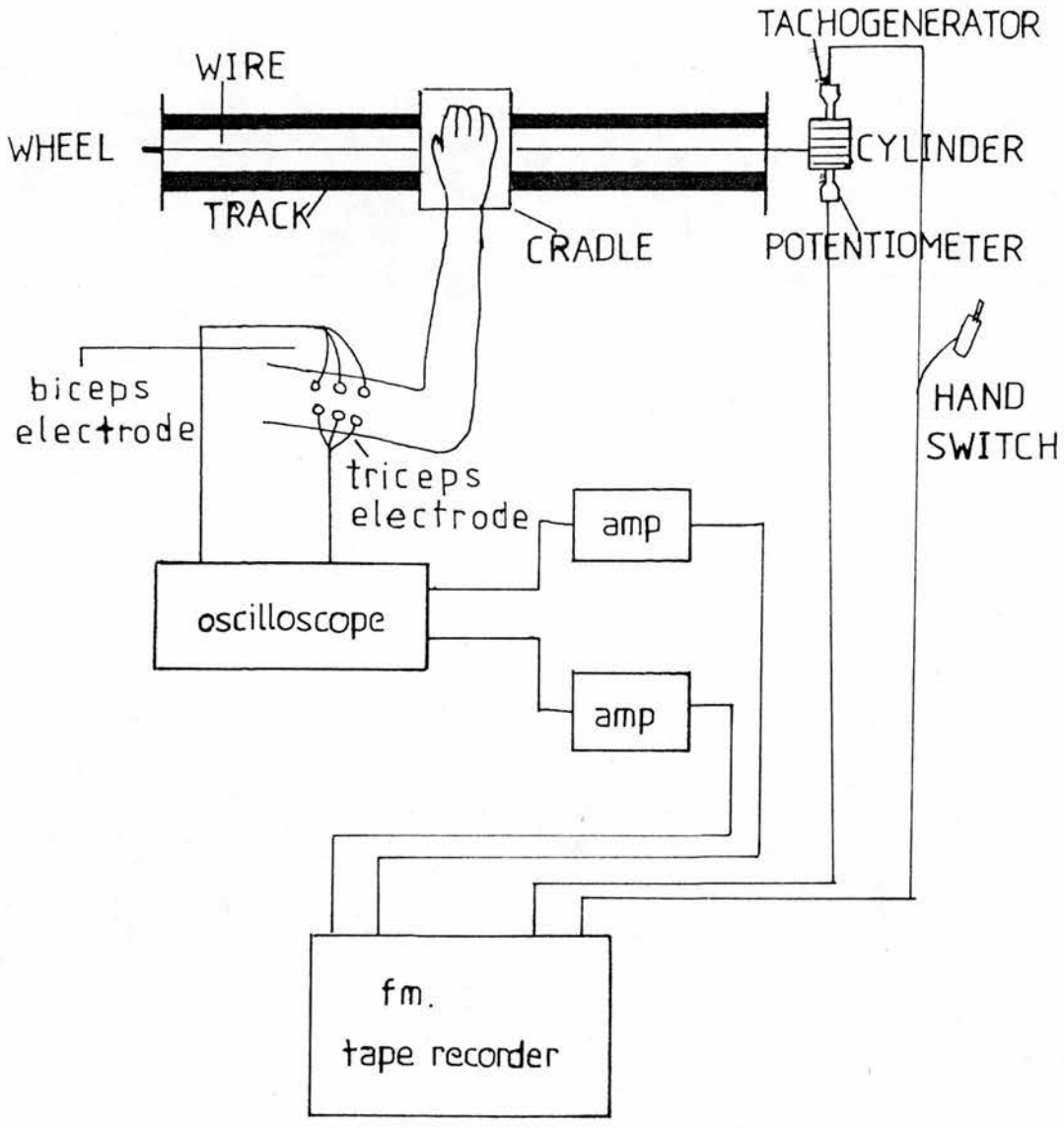



Figure 3.1.2 Recording set-up.

Figure 3.1.3 - The experimental set-up with vision occluded.

target (20 cm).

A padded, plastic wrist cradle was constructed to constrain the hand to the track. A thin strip of metal arched over the top of the cradle. At its acme was a bright red pointer, the tip of which ended just below the perspex. The cradle was set on two nylon tubes which fitted around the steel rods and permitted the cradle to move smoothly, without obstruction, along the length of the track. A strand of cable looped along the length of the track, between the rods and was attached at both ends to either side of the cradle. At one end of the track the cable wound several times round a brass cylinder into a close-fitting, etched spiral. At the other end it looped over a small brass wheel. Firmly attached to the axes of the cylinder was; on one side, the shaft of a tachogenerator and on the other, the shaft of a potentiometer, which was in circuit with a voltage stabiliser connected to two 9V batteries. Therefore, when the cradle was moved the output voltages from the tachogenerator and potentiometer reflected respectively, its speed and position on the track. Error of measure for the potentiometer was estimated at +/- 0.5% of the full range. The voltages were fed into two channels of a Racal four channel FM tape recorder. Also connected to the tachogenerator channel was a switch linked to a 9V battery. When depressed and released it momentarily sent a pulse to the tape recorder. This also emitted a loud

click, so it was used as the signal to commence movement. This point on the tape could subsequently be identified by the spike in the record. The other two channels of the tape recorder received EMG data from stainless steel electrodes. Enroute, the signals were displayed on an oscilloscope. The EMG signals were amplified one thousand times and filtered with a bandwidth of 20Hz to 2 KHz, prior to recording.

A seat with a straight back and arm rests was provided for the subject. In the FV conditions the perspex permitted sight of the relationship between the pointer and the target. NV conditions (as illustrated in Figure 3.1.3) were created by placing a piece of black cardboard on top of the perspex such that target strips on its surface were directly over those fixed to the perspex. In order that the position of the pointer could not be inferred from arm position, a cloak was worn by the subject. The hem was attached to the wooden frame of the assembly and a taut apron was formed by draping the cloak over a vertical rod clamped to the chair arm-rest furthest from the table. All sight of arm movement was thus denied.

Procedure

The experiment was conducted in a treatment room of the hospital's Physiotherapy Department. Due to the time it took to set-up and run each condition and in order to

prevent patient fatigue, the entire experiment was spread over several sessions.

Prior to the subject entering the room the potentiometer was calibrated with respect to measured distances on the track. On arrival, the subject was seated in the chair which was then positioned for optimal flexion/extension of the elbow. The angle of tilt of the frame was also adjusted to allow for subject's body height and thus permit adequate view of the cradle and track. Surface electrodes were placed on the bellies of the triceps and biceps following cleaning with methylated spirits. Placement and contact was checked by asking the subject to attempt maximum contractions in the directions of flexion and extension against resistance and observing the oscilliscope traces. The hand and wrist were then placed in the cradle, with the fingers semi-flexed around a grip.

Practice trials were conducted to acquaint the subject with the task. They also served as a training period, wherein he attempted to relax his muscles prior to movement and restrict movement to about the elbow joint. After this, if the condition was to be an NV one, the cardboard and cloak screens were employed. Resting levels of the muscles at the start position were then sampled.

In performing the experimental trials the subject was instructed not to "throw" his arm at the target, but to move as quickly as possible and line up as accurately as possible underneath the target strip. He was told that if he overshot he should not attempt to correct for it. It was emphasised that the arm should be relaxed prior to movement. Before each trial a verbal "ready" signal was given, accompanied by statement of the target distance; "long" or "short". There was then a variable foreperiod of up to six seconds during which the oscilloscope was monitored for increased EMG activity. The handswitch was then depressed and released producing a loud click which was the signal to commence movement. The trial was considered over when the patient judged himself to have lined the pointer up underneath the target strip. The cradle and arm were then moved back to the start by the experimenter. If the patient moved to the wrong target, or used noticeable trunk or shoulder movement, the trial was repeated.

Data Analysis

The tape recorder and tapes were transported to the Psychology Department where the experimental results were digitised and stored using a PDP-11/34 computer. Enroute to the computer's A/D converters, the EMG channnels were full wave rectified and integrated with a time constant of 125 msecs. The data was stored as a separate file for each condition, in sets of four records. Each set

contained the information from one trial, with each record corresponding with a tape recorder channel. Analysis was on a record by record basis using a cursor and display generated on a VT11 graphics terminal. Prior to analysis, the calibration and EMG resting levels were sampled. The extraction of data and some subsequent calculation, resulted in the following measures:-

1) Constant error (CE) - The accuracy of the movement with respect to the target's position, taking the sign of the error into account.

2) Initiation time (IT) - The time it took the subject to respond to the signal to move and start moving.

3) Movement time (MT) - The time from starting to move to completing the action.

(In obtaining 2) and 3), the start and end of the movement were defined from the position and velocity records. Movement was judged to have commenced when velocity exceeded 2.5cm/sec and if the first submovement was greater than or equal to 5% of the total distance covered by the eventual trajectory. Similar criteria were applied in defining the cessation of movement.)

4) Number of submovements (NSM) - The smoothness of the movement expressed in terms of the number of velocity peaks.

5) Muscle activity - The percentage of the movement time during which a) the biceps and b) the triceps were active, as defined by voltage levels greater than two standard deviations above mean resting level.

Analysis of these measures was conducted individually for each subject using four-way, 2 x 2 x 2 x 2, (Arm x Direction x Vision x Distance) independent measures ANOVA's. The normal subject's data was analysed using a three-way, 2 x 2 x 2, (Direction x Vision x Distance) independent ANOVA. Post-hoc comparison of individual means was conducted using a related t test.

## Results

In presenting the results, the measures are ordered according to the list on page 160.

Since CE was one of the principal measures in the previous Chapter it is examined first to assess subjects' accuracy of performance. IT and MT measures are then assessed in order to determine any abnormalities in the time it takes the patient to prepare and execute movement. Next, attention is turned to the execution phase of the task, with analysis of the velocity profiles of the movements and also the underlying EMG activity. Assessment of how these last two measures correlate was considered but not conducted. This was principally because there was always the possibility of joints other than the elbow making some contribution to the velocity profile. Another argument against correlating these measures has been advanced by Cooke (1980). Cooke found highly non-significant correlations and attributed this to high variability in EMG. burst, magnitude, and duration across trials.

### Constant Error

Means and standard deviations of constant or signed error are tabulated for each subject in Table 3.1.2, with the means illustrated in Figure 3.1.4. The ANOVA results are presented in Table 3.1.3. As can be seen from the Figure, the normal subject (AL) demonstrated highly accurate performance across all experimental conditions

Table 3.1.2
Mean (S.D.) Constant Error

| CONDITIONS | | | SUBJECTS EW | JG | MS | WI | AL |
|---|---|---|---|---|---|---|---|
| | E V | L | +3.22 | -0.20 | +1.44 | +2.68 | -0.04 |
| | | | (0.99) | (0.60) | (1.15) | (0.55) | (0.51) |
| | | S | +1.86 | +0.94 | +1.01 | +1.42 | +0.28 |
| | | | (1.57) | (0.95) | (0.96) | (0.74) | (0.74) |
| | NV | L | +0.55 | +0.21 | +1.17 | -0.27 | -0.46 |
| | | | (1.94) | (1.55) | (1.91) | (1.70) | (2.55) |
| UA | | S | +0.90 | +0.29 | +1.58 | +0.57 | +0.35 |
| | | | (1.01) | (1.26) | (1.24) | (0.66) | (1.76) |
| | F V | L | +1.22 | -0.20 | +0.68 | +0.17 | +0.66 |
| | | | (1.70) | (0.81) | (0.92) | (0.35) | (0.60) |
| | | S | +0.89 | +0.09 | +0.82 | +0.85 | +0.66 |
| | | | (0.91) | (1.11) | (0.85) | (0.52) | (0.41) |
| | NV | L | +0.50 | +0.43 | +0.85 | -0.58 | -0.36 |
| | | | (3.05) | (2.27) | (2.21) | (0.68) | (2.29) |
| | | S | +1.07 | -0.30 | +0.48 | -0.32 | +0.28 |
| | | | (1.40) | (1.14) | (1.25) | (0.56) | (1.75) |
| | E V | L | -1.90 | -0.09 | +1.23 | +2.39 | |
| | | | (0.86) | (1.50) | (1.19) | (0.39) | |
| | | S | -0.90 | -0.26 | +0.47 | +2.94 | |
| | | | (1.70) | (1.00) | (1.21) | (0.55) | |
| | NV | L | -7.98 | -7.55 | -5.35 | -8.45 | |
| | | | (2.26) | (2.81) | (2.87) | (1.18) | |
| AA | | S | -4.67 | -2.61 | -0.73 | -2.12 | |
| | | | (2.22) | (0.81) | (0.55) | (0.66) | |
| | F V | L | -0.77 | -0.08 | -1.93 | -2.09 | |
| | | | (1.03) | (1.75) | (1.76) | (1.85) | |
| | | S | +2.80 | +1.07 | +0.24 | -0.06 | |
| | | | (0.73) | (0.70) | (0.78) | (1.21) | |
| | NV | L | +1.08 | +0.64 | +0.73 | +1.49 | |
| | | | (1.81) | (1.57) | (1.02) | (0.69) | |
| | | S | +3.51 | +0.61 | +0.70 | +1.71 | |
| | | | (2.19) | (1.69) | (1.38) | (0.88) | |

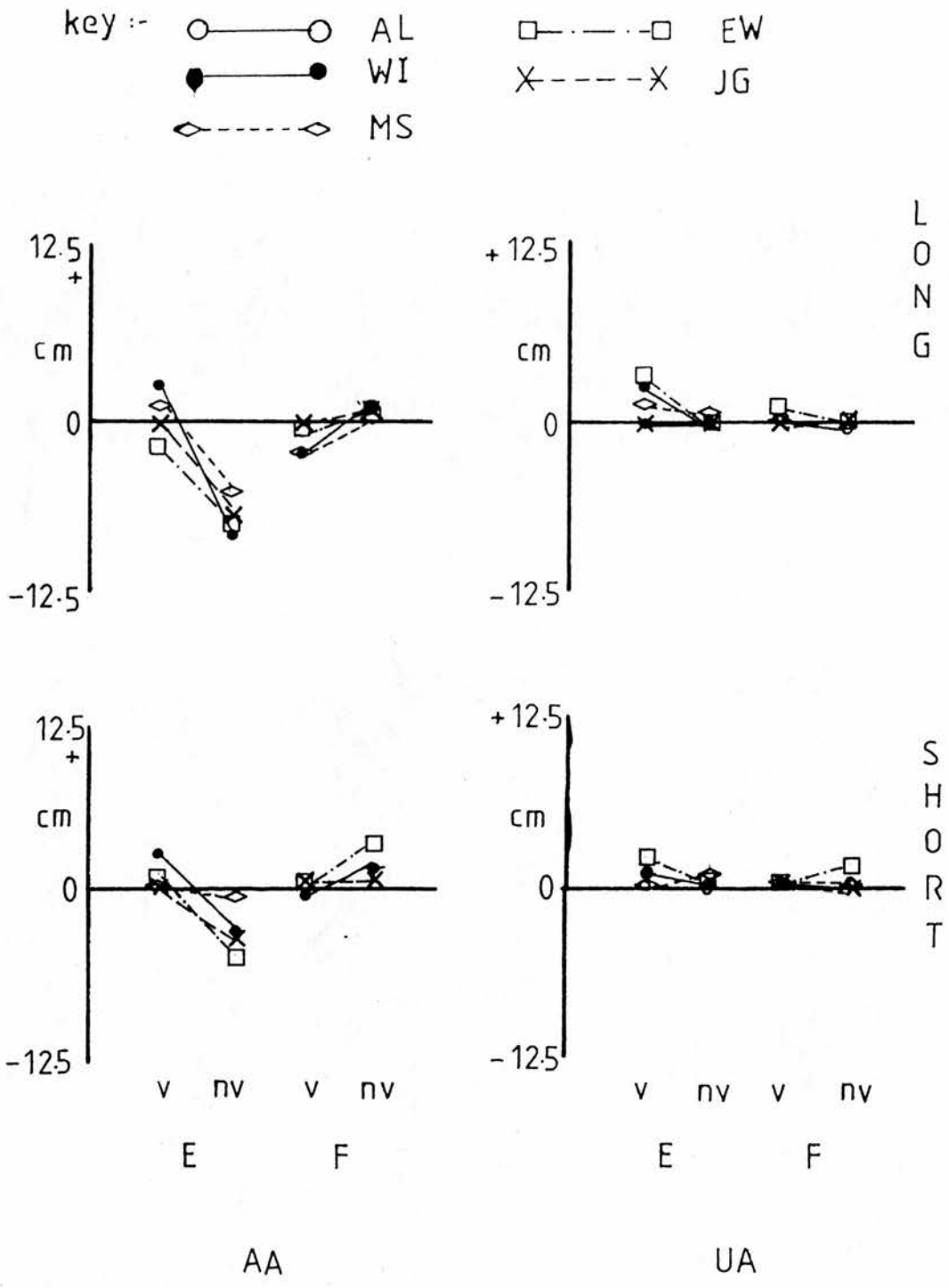



Figure 3.1.4 MEAN CONSTANT ERROR ( cm )

Table 3.1.3

ANOVA Results for Constant Error

| SOURCE OF VARIATION | MS | SUBJECTS EW | JG | WI |
|---|---|---|---|---|
| ARM | 47.866 *** | 83.969 *** | 26.038 *** | 55.198 *** |
| DIR | 0.913 ns | 47.728 *** | 38.211 *** | 2.940 ns |
| VIS | 6.119 * | 24.877 *** | 26.148 *** | 192.602 *** |
| DIST | 9.858 ** | 16.784 *** | 13.217 *** | 67.755 *** |
| ARM X DIR | 12.559 *** | 91.264 *** | 57.353 *** | 81.290 *** |
| ARM X VIS | 6.841 ** | 1.250 ns | 26.926 *** | 16.974 *** |
| DIR X VIS | 32.437 *** | 84.651 *** | 32.815 *** | 389.256 *** |
| ARM X DIST | 11.647 *** | 23.150 *** | 7.391 ** | 53.938 *** |
| DIR X DIST | 1.111 ns | 0.416 ns | 7.989 ** | 7.780 ** |
| VIS X DIST | 3.674 ns | 4.340 * | 0.922 ns | 23.226 *** |
| ARM X DIR X VIS | 38.547 *** | 40.110 *** | 26.369 *** | 272.988 *** |
| ARM X DIR X DIST | 0.680 ns | 3.258 ns | 1.235 ns | 26.106 *** |
| ARM X VIS X DIST | 2.422 ns | 0.115 ns | 10.755 ** | 3.816 ns |
| DIR X VIS X DIST | 23.778 *** | 0.001 ns | 11.621 *** | 74.368 *** |
| ARM X DIR X VIS X DIST | 11.573 *** | 0.615 ns | 11.402 *** | 18.703 *** |

| | |
|---|---|
| ns | non-significant |
| * | $p < 0.05$ |
| ** | $p < 0.01$ |
| *** | $p < 0.001$ |

with no significant differences resulting from the three-way ANOVA. In general the patients' unaffected arm (UA) performance looks to be only marginally less accurate than normal. Across the conditions, post-hoc tests demonstrate little effect of the other factors on the UA.

Although the presence of a main effect of Arm in all patients demonstrates that the affected arm (AA) is significantly less accurate than the UA, the principal reason for this is the trend for all patients to undershoot the target when performing extension under the no vision (NV) condition. Figure 3.1.4 illustrates this quite clearly. Post-hoc analysis of all three interactions, Arm x Dir, Arm x Vis and Arm x Dir x Vis, present in total for all subjects (except for EW), reveals this to be highly significant. The lack of the Arm x Vis interaction for EW is explained by the increase in accuracy under the NV condition for UA extension.

Another significance of these interactions is the tendency for loss of vision in AA flexion to result in overshoot beyond the level of accuracy found under full vision (FV). This is significant in three of the four patients (EW, MS and WI).

Both these results however, need to be considered in the light of significant interactions involving the

factor distance (Dist). Significant main effects of Dist, found for all patients, is due to movements aimed at the shorter target being more accurate. The presence of significant Arm x Dist interactions narrows this result down to the AA. The primary influence of this factor emerges from post-hoc scrutiny of the significant Dir x Vis x Dist and four-way interactions found. In all patients, the effect of withdrawal of vision on AA extension was greater for the longer target distance. With flexion the results are less clear.

Withdrawal of vision therefore only affects the accuracy of the AA movement and primarily those attempting the greater target distance. In extension loss of vision results in an undershoot, whilst in flexion the trend is in the opposite direction towards an overshoot.

Initiation Time

Means and standard deviations of initiation time (IT) are presented in Table 3.1.4 with the means plotted in Figure 3.1.5 for each subject. The ANOVA results are listed in Table 3.1.5. As the Figure shows, IT is higher than normal for both the UA and AA. This is the only consistent result to emerge from this measure. Analysis of AL's data revealed only one point of note. A significant main effect of Vis indicated that with the normal arm, withdrawal of vision elevated IT.

Table 3.1.4
Mean (S.D.) Initiation Time (secs)

| CONDITIONS | | | SUBJECTS EW | JG | MS | WI | AL |
|---|---|---|---|---|---|---|---|
| | E V | L | 0.475 | 0.435 | 0.444 | 0.568 | 0.231 |
| | | | (0.158) | (0.120) | (0.160) | (0.094) | (0.052) |
| | | S | 0.474 | 0.448 | 0.383 | 0.536 | 0.238 |
| | | | (0.160) | (0.147) | (0.171) | (0.158) | (0.058) |
| | NV | L | 0.486 | 0.505 | 0.449 | 0.339 | 0.335 |
| | | | (0.105) | (0.224) | (0.168) | (0.171) | (0.075) |
| UA | | S | 0.452 | 0.554 | 0.427 | 0.451 | 0.333 |
| | | | (0.197) | (0.214) | (0.134) | (0.208) | (0.081) |
| | F V | L | 0.511 | 0.364 | 0.614 | 0.472 | 0.229 |
| | | | (0.234) | (0.137) | (0.209) | (0.136) | (0.083) |
| | | S | 0.596 | 0.402 | 0.608 | 0.509 | 0.222 |
| | | | (0.300) | (0.110) | (0.248) | (0.191) | (0.069) |
| | NV | L | 0.618 | 0.480 | 0.578 | 0.546 | 0.309 |
| | | | (0.249) | (0.196) | (0.217) | (0.247) | (0.061) |
| | | S | 0.634 | 0.532 | 0.458 | 0.508 | 0.294 |
| | | | (0.226) | (0.178) | (0.166) | (0.184) | (0.104) |
| | E V | L | 0.656 | 0.390 | 0.884 | 0.498 | |
| | | | (0.232) | (0.206) | (0.174) | (0.102) | |
| | | S | 0.623 | 0.321 | 0.757 | 0.481 | |
| | | | (0.205) | (0.115) | (0.255) | (0.106) | |
| | NV | L | 1.018 | 0.438 | 0.798 | 0.469 | |
| | | | (0.151) | (0.130) | (0.150) | (0.112) | |
| AA | | S | 0.961 | 0.463 | 0.827 | 0.561 | |
| | | | (0.185) | (0.223) | (0.184) | (0.277) | |
| | F V | L | 0.629 | 0.400 | 0.590 | 0.376 | |
| | | | (0.142) | (0.094) | (0.262) | (0.195) | |
| | | S | 0.722 | 0.360 | 0.580 | 0.404 | |
| | | | (0.165) | (0.079) | (0.173) | (0.139) | |
| | NV | L | 0.542 | 0.388 | 0.684 | 0.382 | |
| | | | (0.185) | (0.074) | (0.111) | (0.153) | |
| | | S | 0.600 | 0.411 | 0.652 | 0.344 | |
| | | | (0.251) | (0.115) | (0.126) | (0.157) | |

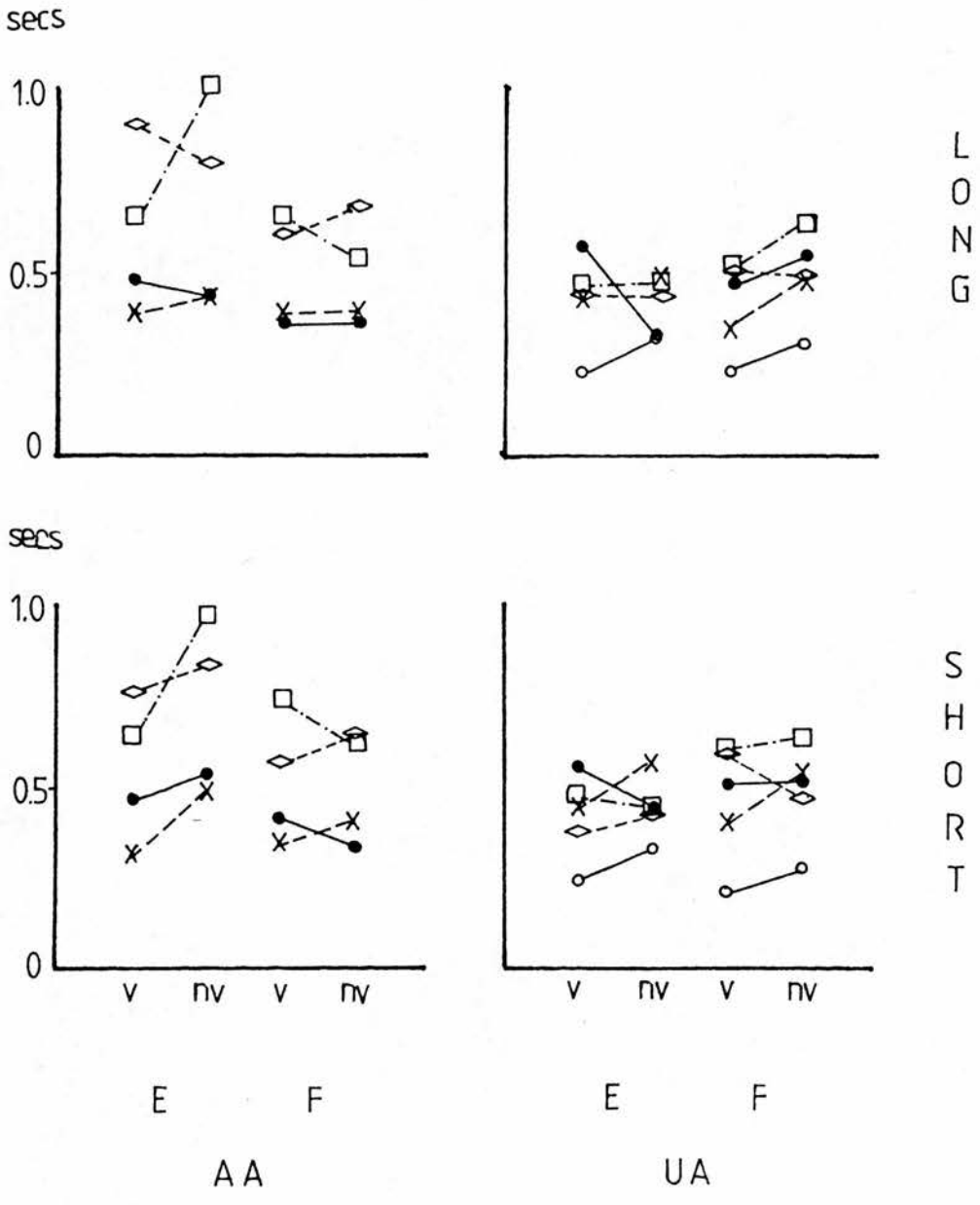



Figure 3.1.5 MEAN INITIATION TIME ( secs )

Table 3.1.5
ANOVA Results for Initiation Time

| SOURCE OF VARIATION | MS | SUBJECTS EW | JG | WI |
|---|---|---|---|---|
| ARM | 58.436 *** | 34.510 *** | 7.712 ** | 3.604 ns |
| DIR | 0.749 ns | 1.380 ns | 1.205 ns | 2.756 ns |
| VIS | 0.003 ns | 5.951 * | 10.844 *** | 1.252 ns |
| DIST | 2.170 ns | 0.246 ns | 0.212 ns | 0.436 ns |
| ARM X DIR | 30.810 *** | 23.313 *** | 0.315 ns | 8.722 ** |
| ARM X VIS | 1.468 ns | 1.942 ns | 0.953 ns | 1.191 ns |
| DIR X VIS | 0.050 ns | 8.639 ** | 0.168 ns | 1.672 ns |
| ARM X DIST | 0.085 ns | 0.000 ns | 1.161 ns | 0.004 ns |
| DIR X DIST | 0.003 ns | 2.165 ns | 0.077 ns | 0.579 ns |
| VIS X DIST | 0.062 ns | 0.395 ns | 1.096 ns | 0.264 ns |
| ARM X DIR X VIS | 3.098 ns | 17.281 *** | 1.250 ns | 5.090 * |
| ARM X DIR X DIST | 0.175 ns | 0.168 ns | 0.000 ns | 0.000 ns |
| ARM X VIS X DIST | 0.778 ns | 0.028 ns | 0.293 ns | 0.014 ns |
| DIR X VIS X DIST | 1.952 ns | 0.034 ns | 0.072 ns | 3.265 ns |
| ARM X DIR X VIS X DIST | 0.011 ns | 0.010 ns | 0.002 ns | 0.041 ns |

| | |
|---|---|
| ns | non-significant |
| * | $p < 0.05$ |
| ** | $p < 0.01$ |
| *** | $p < 0.001$ |

As can be seen from Table 3.1.5, this result is not generally reflected in either UA or AA performance across the hemiparetic subjects. Only two of these subjects show main effects of Vis (EW and JG). With no interactions present, JG is the only patient to follow the results of AL. The main effect for EW is qualified by Dir x Vis and Arm x Dir x Vis interactions which together indicate that the only significant effect loss of vision has upon IT is to raise it prior to extension of the AA. MS shows no significant effects of Vis whatsoever and WI shows a significant decrease in IT across all movements when vision is withdrawn.

Two subjects exhibit IT to be faster for the AA (JG and WI). However this is only a main effect for JG. The lack of main effect of Vis for WI can be understood by analysis of the significant Arm x Dir and Arm x Dir x Vis interactions. The arms differ only on flexion and although IT in AA extension is significantly higher than AA flexion, a non-significant result, in the opposite direction, for the UA cancels out a significant difference between the arms at the level of main effect. Contrary to this, MS and EW show main effects in the opposite direction which are qualified by interactions. Both have significant Arm x Dir interactions which are due to the arms only differing on extension.

Movement Time

One of the variables which determines movement time (MT)

is the task distance (Fitts, 1954). Since the present experimental set-up consisted of two target distances, with one being twice as far as the other, a lawful distinction for Dist on MT might be expected. However, following from the results on accuracy this may be confounded by the degree of error in some of the long distance conditions. Therefore a more appropriate measure to analyse speed of movement is velocity. This was calculated on a trial by trial basis, by dividing the distance moved (target distance plus CE) by the MT (Table 3.1.6).

Velocity - Means and standard deviations of the average velocity attained across the task movements are presented in Table 3.1.7, with the means plotted in Figure 3.1.6, and the results of individual ANOVA's listed in Table 3.1.8. The Figure shows that for patients both limbs moved with a lower mean velocity than normal. For the normal subject, the three-way ANOVA revealed a main effect of Vis and one of Dist. Fig 3.1.6 shows that velocity decreases when vision is withdrawn. In addition its value is not as great when the movement is to the nearer target. Qualifying these, there is a Vis x Dist interaction wherein the effect of Dist is greater under FV conditions.

The significant Arm x Dist interactions found for all patients, indicate in three of them (EW, JG and WI)

Table 3.1.6
Mean (S.D.) Movement Time (secs)

| CONDITIONS | | | SUBJECTS EW | JG | MS | WI | AL |
|---|---|---|---|---|---|---|---|
| UA | E | V L | 2.141 | 0.921 | 1.610 | 1.877 | 0.853 |
| | | | (0.634) | (0.115) | (0.353) | (0.338) | (0.196) |
| | | S | 1.584 | 0.697 | 1.145 | 0.974 | 0.669 |
| | | | (0.377) | (0.169) | (0.229) | (0.156) | (0.133) |
| | NV | L | 2.737 | 1.038 | 1.229 | 1.523 | 1.118 |
| | | | (0.579) | (0.245) | (0.366) | (0.318) | (0.199) |
| | | S | 2.020 | 0.769 | 0.901 | 1.178 | 0.767 |
| | | | (0.601) | (0.109) | (0.260) | (0.250) | (0.149) |
| | F | V L | 1.855 | 1.023 | 1.992 | 1.530 | 0.732 |
| | | | (0.404) | (0.227) | (0.508) | (0.157) | (0.161) |
| | | S | 1.342 | 0.797 | 1.407 | 1.023 | 0.586 |
| | | | (0.345) | (0.242) | (0.365) | (0.080) | (0.071) |
| | NV | L | 3.080 | 1.203 | 1.540 | 1.172 | 1.047 |
| | | | (0.494) | (0.323) | (0.474) | (0.125) | (0.138) |
| | | S | 1.745 | 0.608 | 1.241 | 0.931 | 0.821 |
| | | | (0.465) | (0.181) | (0.222) | (0.151) | (0.111) |
| AA | E | V L | 7.096 | 1.661 | 3.636 | 2.493 | |
| | | | (1.570) | (0.543) | (1.034) | (0.417) | |
| | | S | 3.970 | 0.925 | 1.881 | 1.200 | |
| | | | (0.940) | (0.362) | (0.622) | (0.139) | |
| | NV | L | 10.312 | 2.037 | 2.625 | 2.098 | |
| | | | (4.986) | (0.409) | (1.171) | (0.219) | |
| | | S | 7.729 | 1.387 | 2.462 | 1.827 | |
| | | | (5.083) | (0.295) | (0.879) | (0.596) | |
| | F | V L | 2.616 | 1.798 | 1.433 | 1.463 | |
| | | | (0.335) | (0.612) | (0.461) | (0.202) | |
| | | S | 2.048 | 1.470 | 0.935 | 1.097 | |
| | | | (0.444) | (0.518) | (0.241) | (0.382) | |
| | NV | L | 2.614 | 1.255 | 1.579 | 1.910 | |
| | | | (0.723) | (0.260) | (0.317) | (0.727) | |
| | | S | 2.061 | 0.846 | 1.047 | 1.402 | |
| | | | (0.555) | (0.312) | (0.179) | (0.279) | |

Table 3.1.7
Mean (S.D.) Velocity (cm/sec)

| CONDITIONS | | | SUBJECTS EW | JG | MS | WI | AL |
|---|---|---|---|---|---|---|---|
| | E V | L | 11.50 | 21.80 | 13.90 | 12.47 | 24.61 |
| | | | (2.63) | (2.70) | (2.97) | (2.41) | (6.05) |
| | | S | 7.87 | 16.31 | 10.04 | 11.93 | 15.86 |
| | | | (1.97) | (3.08) | (2.38) | (1.68) | (3.01) |
| | NV | L | 7.78 | 20.36 | 18.62 | 13.40 | 18.15 |
| | | | (1.56) | (4.47) | (5.56) | (2.78) | (4.92) |
| UA | | S | 5.72 | 13.56 | 14.13 | 9.44 | 14.00 |
| | | | (1.44) | (2.28) | (5.49) | (2.65) | (3.80) |
| | F V | L | 11.91 | 20.24 | 10.97 | 13.32 | 29.21 |
| | | | (2.72) | (5.00) | (2.59) | (1.30) | (5.12) |
| | | S | 8.60 | 13.92 | 8.02 | 10.70 | 18.46 |
| | | | (2.14) | (4.63) | (1.43) | (1.39) | (2.29) |
| | NV | L | 6.81 | 18.02 | 15.06 | 16.74 | 19.04 |
| | | | (1.47) | (4.48) | (6.03) | (1.63) | (3.43) |
| | | S | 6.71 | 16.99 | 8.58 | 10.65 | 12.65 |
| | | | (1.62) | (4.19) | (1.10) | (1.90) | (2.20) |
| | E V | L | 2.70 | 13.02 | 6.39 | 9.21 | |
| | | | (0.80) | (3.88) | (2.42) | (1.57) | |
| | | S | 2.89 | 12.03 | 6.32 | 10.93 | |
| | | | (0.80) | (4.53) | (2.67) | (1.39) | |
| | NV | L | 1.68 | 6.30 | 6.48 | 5.57 | |
| | | | (1.23) | (1.88) | (2.63) | (0.87) | |
| AA | | S | 1.10 | 5.51 | 4.11 | 4.72 | |
| | | | (0.94) | (1.24) | (1.18) | (1.51) | |
| | F V | L | 7.46 | 12.52 | 13.90 | 12.39 | |
| | | | (0.98) | (4.90) | (4.71) | (1.64) | |
| | | S | 5.28 | 8.50 | 12.07 | 9.88 | |
| | | | (1.29) | (3.22) | (5.28) | (2.91) | |
| | NV | L | 8.70 | 16.96 | 13.70 | 12.33 | |
| | | | (2.74) | (3.20) | (3.38) | (3.38) | |
| | | S | 7.00 | 14.97 | 10.56 | 8.63 | |
| | | | (2.19) | (7.77) | (2.51) | (1.58) | |

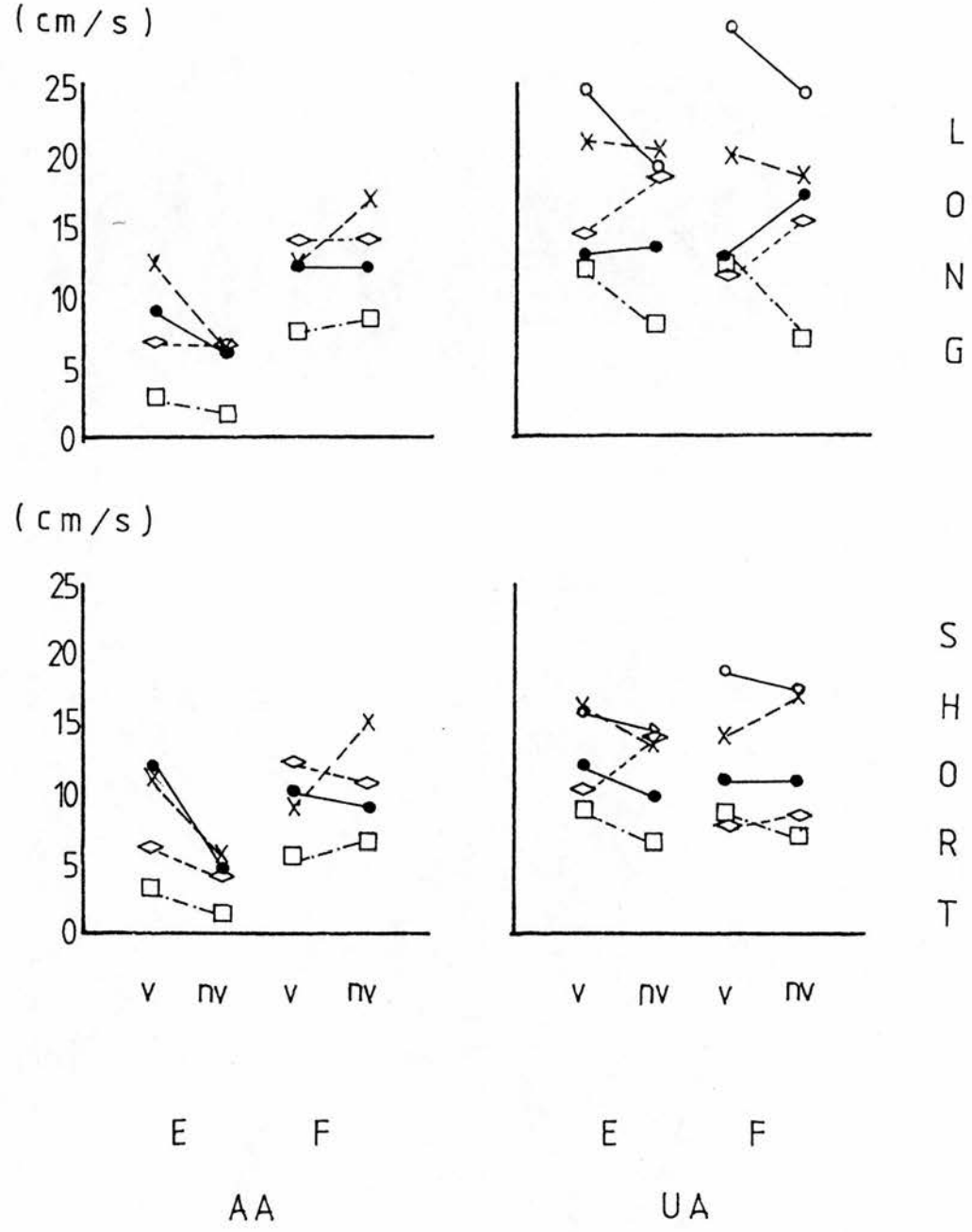



Figure 3.1.6 MEAN VELOCITY ( cm/s )

Table 3.1.8
ANOVA Results for Velocity

| SOURCE OF VARIATION | MS | SUBJECTS EW | JG | WI |
|---|---|---|---|---|
| ARM | 31.519 *** | 178.455 *** | 97.195 *** | 94.940 *** |
| DIR | 7.849 ** | 88.835 *** | 6.442 * | 43.781 *** |
| VIS | 4.395 * | 31.840 *** | 1.183 ns | 13.291 *** |
| DIST | 30.069 *** | 35.233 *** | 27.691 *** | 52.313 *** |
| ARM X DIR | 72.620 *** | 70.480 *** | 13.216 *** | 11.322 *** |
| ARM X VIS | 14.166 *** | 33.361 *** | 0.038 ns | 25.574 *** |
| DIR X VIS | 0.666 ns | 4.262 * | 31.358 *** | 27.584 *** |
| ARM X DIST | 5.096 * | 4.598 * | 5.168 * | 9.416 ** |
| DIR X DIST | 0.618 ns | 0.289 ns | 0.019 ns | 19.378 *** |
| VIS X DIST | 2.861 ns | 3.974 ns | 1.419 ns | 17.243 *** |
| ARM X DIR X VIS | 0.990 ns | 9.357 ** | 13.440 *** | 1.981 ns |
| ARM X DIR X DIST | 0.100 ns | 6.562 * | 3.095 ns | 1.252 ns |
| ARM X VIS X DIST | 0.014 ns | 5.066 * | 0.133 ns | 1.489 ns |
| DIR X VIS X DIST | 0.173 ns | 1.646 ns | 2.615 ns | 0.269 ns |
| ARM X DIR X VIS X DIST | 0.717 ns | 0.030 ns | 0.837 ns | 0.311 ns |

ns non-significant
* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

that distance only affects the UA resulting in a lower mean velocity for the shorter target distance. In the fourth subject (MS), significance of this is prevented.

The main effect of Arm present in all the patients' results shows the AA generally moved more slowly than the UA. However the presence of many interactions involving this factor require consideration. All patients have main effects of Dir and significant Arm x Dir interactions. Post-hoc analysis reveals this to be due to a significantly lower mean velocity in the conditions requiring extension of the AA. WI is the only subject to show a similar result with the UA.

As Figure 3.1.6 shows, it is difficult to arrive at a clear conclusion concerning the effects of Vis upon mean velocity of the UA. For the AA, the general pattern is for a decrease in velocity when vision is withdrawn and this is mainly for extension movements. In two of the subjects (EW and JG) there are significant Arm x Dir x Vis interactions to that effect. An additional point from those interactions, is that flexion movements by the AA have a higher mean velocity under NV conditions. In the other two subjects, there is no effect of Vis on flexion of the AA.

Number of Submovements

Means and standard deviations of the number of

Table 3.1.9
Mean (S.D.) Number of Submovements

| CONDITIONS | | | SUBJECTS EW | JG | MS | WI | AL |
|---|---|---|---|---|---|---|---|
| | E | V L | 2.10 | 1.90 | 1.90 | 2.40 | 1.70 |
| | | | (0.88) | (0.57) | (0.67) | (0.70) | (0.53) |
| | | S | 2.30 | 1.40 | 2.00 | 1.40 | 1.50 |
| | | | (0.82) | (0.52) | (0.67) | (0.70) | (0.53) |
| | NV | L | 3.60 | 1.70 | 1.90 | 2.20 | 1.80 |
| | | | (1.08) | (0.67) | (0.88) | (0.63) | (0.63) |
| UA | | S | 2.90 | 1.20 | 1.70 | 2.00 | 1.40 |
| | | | (1.29) | (0.42) | (0.82) | (0.82) | (0.52) |
| | F | V L | 2.60 | 1.60 | 2.80 | 2.20 | 1.20 |
| | | | (1.17) | (0.52) | (0.63) | (0.63) | (0.42) |
| | | S | 2.30 | 1.60 | 2.30 | 1.90 | 1.40 |
| | | | (0.82) | (0.52) | (2.67) | (0.57) | (0.52) |
| | NV | L | 3.20 | 2.30 | 2.10 | 1.60 | 2.00 |
| | | | (1.23) | (0.48) | (0.74) | (0.84) | (0.67) |
| | | S | 2.50 | 1.30 | 2.00 | 1.90 | 2.00 |
| | | | (1.18) | (0.67) | (0.47) | (0.86) | (0.47) |
| | E | V L | 8.70 | 3.50 | 4.60 | 5.20 | |
| | | | (2.83) | (0.97) | (1.43) | (1.23) | |
| | | S | 6.90 | 1.80 | 2.40 | 3.00 | |
| | | | (2.08) | (0.42) | (0.52) | (0.00) | |
| | NV | L | 10.20 | 3.80 | 4.50 | 5.90 | |
| | | | (3.16) | (0.42) | (1.08) | (0.88) | |
| AA | | S | 9.00 | 2.70 | 4.10 | 4.30 | |
| | | | (1.14) | (0.48) | (1.10) | (1.57) | |
| | F | V L | 4.10 | 2.70 | 2.20 | 2.70 | |
| | | | (1.10) | (0.48) | (0.42) | (0.82) | |
| | | S | 4.00 | 2.10 | 1.80 | 1.80 | |
| | | | (0.47) | (0.74) | (0.42) | (0.63) | |
| | NV | L | 4.60 | 2.20 | 3.00 | 4.00 | |
| | | | (1.17) | (0.42) | (0.47) | (1.15) | |
| | | S | 3.80 | 1.70 | 2.00 | 3.30 | |
| | | | (1.62) | (0.48) | (0.47) | (0.67) | |

Table 3.1.10
Mean (S.D.) Submovement Distance

| CONDITIONS | | | SUBJECTS EW | JG | MS | WI | AL |
|---|---|---|---|---|---|---|---|
| UA | E | V L | 13.54 (7.00) | 11.52 (4.30) | 12.51 (4.80) | 11.51 (6.13) | 13.59 (5.42) |
| | | S | 5.78 (2.45) | 8.67 (2.76) | 6.20 (2.51) | 9.46 (3.03) | 7.69 (2.71) |
| | NV | L | 6.27 (2.29) | 13.81 (5.83) | 13.08 (5.51) | 9.34 (1.58) | 12.56 (5.86) |
| | | S | 4.55 (2.25) | 9.31 (2.63) | 8.41 (4.01) | 6.27 (2.86) | 8.11 (2.58) |
| | F | V L | 10.38 (6.26) | 13.80 (5.08) | 7.70 (1.67) | 10.05 (3.65) | 18.58 (4.35) |
| | | S | 5.46 (2.54) | 7.05 (2.75) | 4.94 (0.97) | 6.31 (2.41) | 8.53 (2.76) |
| | NV | L | 8.10 (5.53) | 9.10 (1.46) | 11.66 (5.99) | 14.91 (6.00) | 11.09 (4.78) |
| | | S | 5.41 (2.67) | 8.50 (2.46) | 5.56 (1.93) | 6.28 (2.99) | 5.38 (1.40) |
| AA | E | V L | 2.32 (0.86) | 6.15 (1.96) | 5.22 (2.40) | 4.57 (1.34) | |
| | | S | 1.73 (0.63) | 5.83 (1.96) | 4.58 (1.23) | 4.31 (0.18) | |
| | NV | L | 1.43 (0.95) | 3.33 (0.86) | 3.33 (0.67) | 1.99 (0.39) | |
| | | S | 0.60 (0.19) | 2.82 (0.59) | 2.40 (0.59) | 2.03 (0.66) | |
| | F | V L | 5.10 (1.77) | 7.68 (2.04) | 8.43 (1.50) | 7.74 (4.49) | |
| | | S | 2.60 (0.38) | 6.13 (2.88) | 6.09 (1.93) | 6.19 (2.41) | |
| | NV | L | 4.87 (1.36) | 9.63 (1.67) | 7.09 (1.33) | 5.90 (2.21) | |
| | | S | 3.90 (1.11) | 6.78 (2.35) | 5.61 (1.20) | 3.68 (0.81) | |

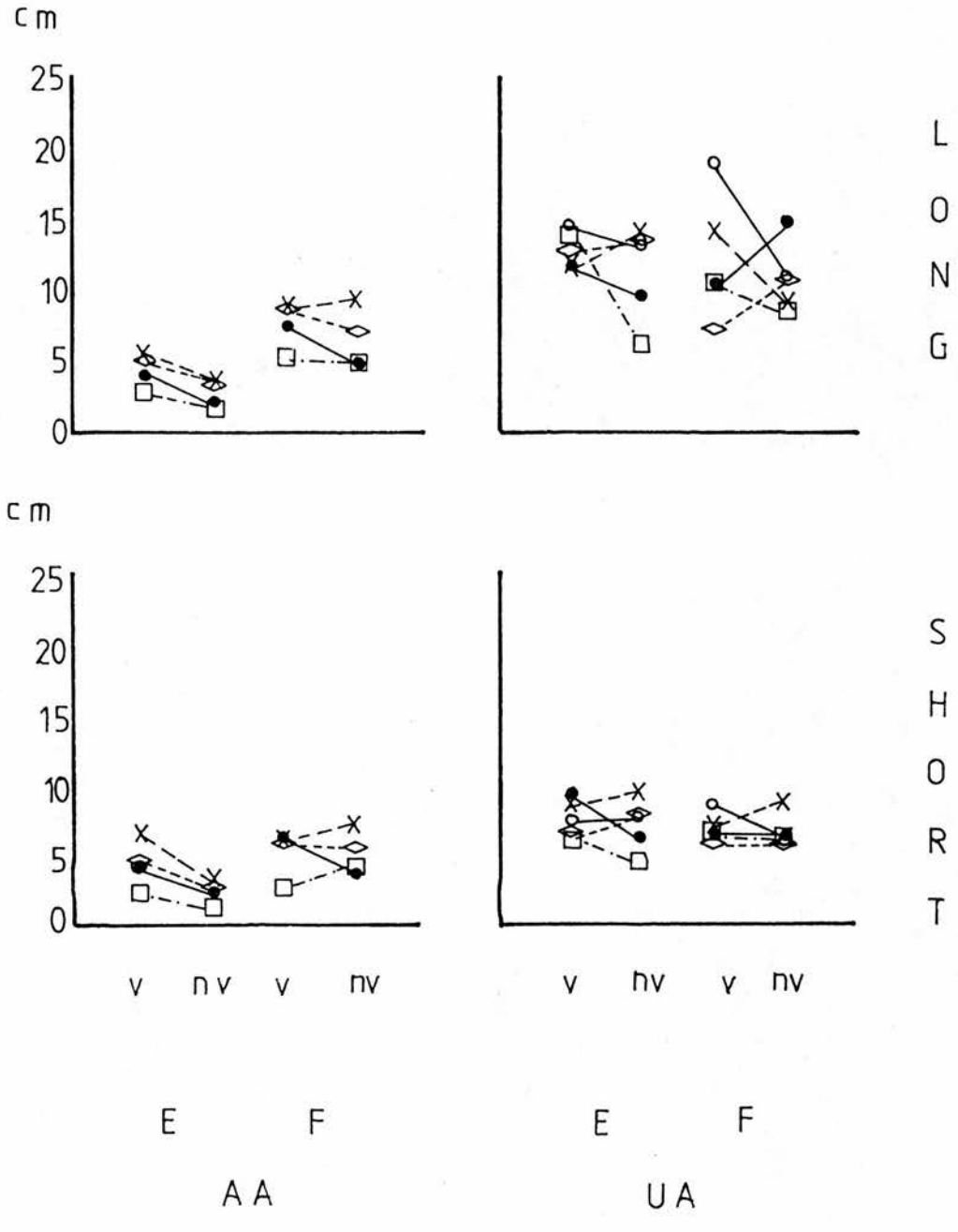



Figure 3.1.7 MEAN SUBMOVEMENT DISTANCE (cm)

Table 3.1.11
ANOVA Results for Mean Submovement Distance

| SOURCE OF VARIATION | MS | SUBJECTS EW | JG | WI |
|---|---|---|---|---|
| ARM | 54.708 *** | 86.821 *** | 80.786 *** | 91.494 *** |
| DIR | 0.134 ns | 5.864 * | 3.783 ns | 8.623 ** |
| VIS | 0.159 ns | 8.829 ** | 0.912 ns | 6.101 * |
| DIST | 46.692 *** | 30.739 *** | 28.747 *** | 29.670 *** |
| ARM X DIR | 35.599 *** | 7.953 ** | 20.793 *** | 5.976 * |
| ARM X VIS | 12.878 *** | 6.211 * | 0.613 ns | 4.845 * |
| DIR X VIS | 1.203 ns | 5.456 * | 0.336 ns | 7.362 ** |
| ARM X DIST | 15.316 *** | 9.470 ** | 6.491 * | 11.720 *** |
| DIR X DIST | 0.001 ns | 0.002 ns | 0.922 ns | 7.501 ** |
| VIS X DIST | 0.094 ns | 5.815 * | 0.656 ns | 2.536 ns |
| ARM X DIR X VIS | 0.015 ns | 0.604 ns | 15.448 *** | 6.026 * |
| ARM X DIR X DIST | 1.401 ns | 0.978 ns | 0.922 ns | 0.880 ns |
| ARM X VIS X DIST | 0.378 ns | 3.100 ns | 2.597 ns | 1.974 ns |
| DIR X VIS X DIST | 1.076 ns | 0.265 ns | 3.239 ns | 1.506 ns |
| ARM X DIR X VIS X DIST | 2.756 ns | 1.981 ns | 5.746 * | 0.541 ns |

| | |
|---|---|
| ns | non-significant |
| * | $p < 0.05$ |
| ** | $p < 0.01$ |
| *** | $p < 0.001$ |

submovements present in task movements are presented in Table 3.1.9. However, as with movement time, the actual distance covered in a trial could affect the number of submovements composing the trajectory. To compensate for this possibility, analysis was conducted on the mean distance traversed by a submovement. these data were calculated by dividing, on a trial by trial basis, the distance covered by the number of submovements present. The means and standard deviations then obtained are presented in Table 3.1.10 with the means illustrated in Figure 3.1.7. Table 3.1.11 contains the ANOVA results for this measure.

Mean Submovement Distance - AL shows a main effect of Dist, with the 10cm target distance having a significantly lesser mean submovement distance. This result also follows for flexion when vision is withdrawn, as evidenced by a main effect of Vis and a significant Vis x Dir interaction.

From Table 3.1.11, it can be seen that all the hemiparetic patients exhibit main effects of Dist. However, for all of them, significant Arm x Dist interactions are present and indicate that the longer mean submovement distance in the long (L) condition are restricted to movements of the UA. In addition, the four main effects of arm, demonstrate the mean submovement distance to be shorter for the AA. This must be qualified with significant Arm x Dir interactions found

for all the patients and due to AA movements in extension having a lower mean submovement distance than in flexion. Further elaboration of this finding results from the presence of significant Arm x Vis interactions for MS, EW and WI and Arm x Dir x Vis interactions for JG and WI. As is evident in Figure 3.1.7, withdrawal of vision principally affects the AA resulting in the lower mean submovement distance found and this is particularly the case for extension.

This result appears to pertain regardless of change in target distance, as only EW has a significant Vis x Dist interaction, with loss of vision only affecting movements to the L target. However, as Figure 3.1.7 shows, this is primarily (although not significantly) due to the differential effect of Vis on extension of the UA. The only other result concerning the factor Vis is the significant four-way interaction found for JG which is due to a lack of effect of Dist for UA flexion under NV conditions.

EMG Activity

Triceps - Means and standard deviations of triceps activity are listed in Table 3.1.12. The means are plotted in Figure 3.1.8 with the ANOVA results presented in Table 3.1.13. AL's data produced only one significant result: a main effect of Dir, which as Figure 3.1.8 demonstrates, results from greater triceps activity

Table 3.1.12
Mean (S.D.) Percentage Triceps Activity

| CONDITIONS | | | SUBJECTS EW | JG | MS | WI | AL |
|---|---|---|---|---|---|---|---|
| | E | V L | 100.00 | 100.00 | 83.24 | 100.00 | 88.30 |
| | | | (0.00) | (0.00) | (34.1) | (0.00) | (14.78) |
| | | S | 100.00 | 100.00 | 91.38 | 100.00 | 85.50 |
| | | | (0.00) | (0.00) | (14.45) | (0.00) | (19.64) |
| | NV | L | 100.00 | 100.00 | 99.75 | 100.00 | 92.70 |
| | | | (0.00) | (0.00) | (0.79) | (0.00) | (6.50) |
| UA | | S | 100.00 | 100.00 | 99.78 | 100.00 | 90.90 |
| | | | (0.00) | (0.00) | (0.67) | (0.00) | (8.28) |
| | F | V L | 17.69 | 27.12 | 19.11 | 22.10 | 32.90 |
| | | | (13.09) | (20.71) | (27.76) | (23.81) | (21.40) |
| | | S | 22.82 | 25.68 | 8.74 | 13.50 | 24.80 |
| | | | (19.05) | (17.58) | (14.24) | (25.15) | (23.79) |
| | NV | L | 19.71 | 31.13 | 11.03 | 26.30 | 26.60 |
| | | | (13.30) | (15.15) | (12.35) | (25.60) | (10.99) |
| | | S | 14.75 | 26.63 | 8.40 | 17.00 | 21.00 |
| | | | (11.94) | (18.51) | (15.10) | (23.48) | (12.72) |
| | E | V L | 100.00 | 96.43 | 94.33 | 100.00 | |
| | | | (0.00) | (7.20) | (9.99) | (0.00) | |
| | | S | 100.00 | 99.48 | 96.30 | 100.00 | |
| | | | (0.00) | (1.64) | (6.34) | (0.00) | |
| | NV | L | 100.00 | 96.60 | 96.16 | 98.29 | |
| | | | (0.00) | (6.05) | (6.93) | (5.41) | |
| AA | | S | 100.00 | 96.33 | 97.92 | 100.00 | |
| | | | (0.00) | (5.17) | (4.58) | (0.00) | |
| | F | V L | 7.51 | 14.86 | 4.07 | 7.38 | |
| | | | (9.93) | (17.61) | (12.87) | (16.26) | |
| | | S | 3.22 | 5.56 | 4.96 | 7.40 | |
| | | | (8.18) | (11.99) | (10.88) | (12.69) | |
| | NV | L | 0.00 | 6.92 | 0.00 | 3.15 | |
| | | | (0.00) | (15.03) | (0.00) | (9.96) | |
| | | S | 0.00 | 3.98 | 0.00 | 2.74 | |
| | | | (0.00) | (12.59) | (0.00) | (8.66) | |

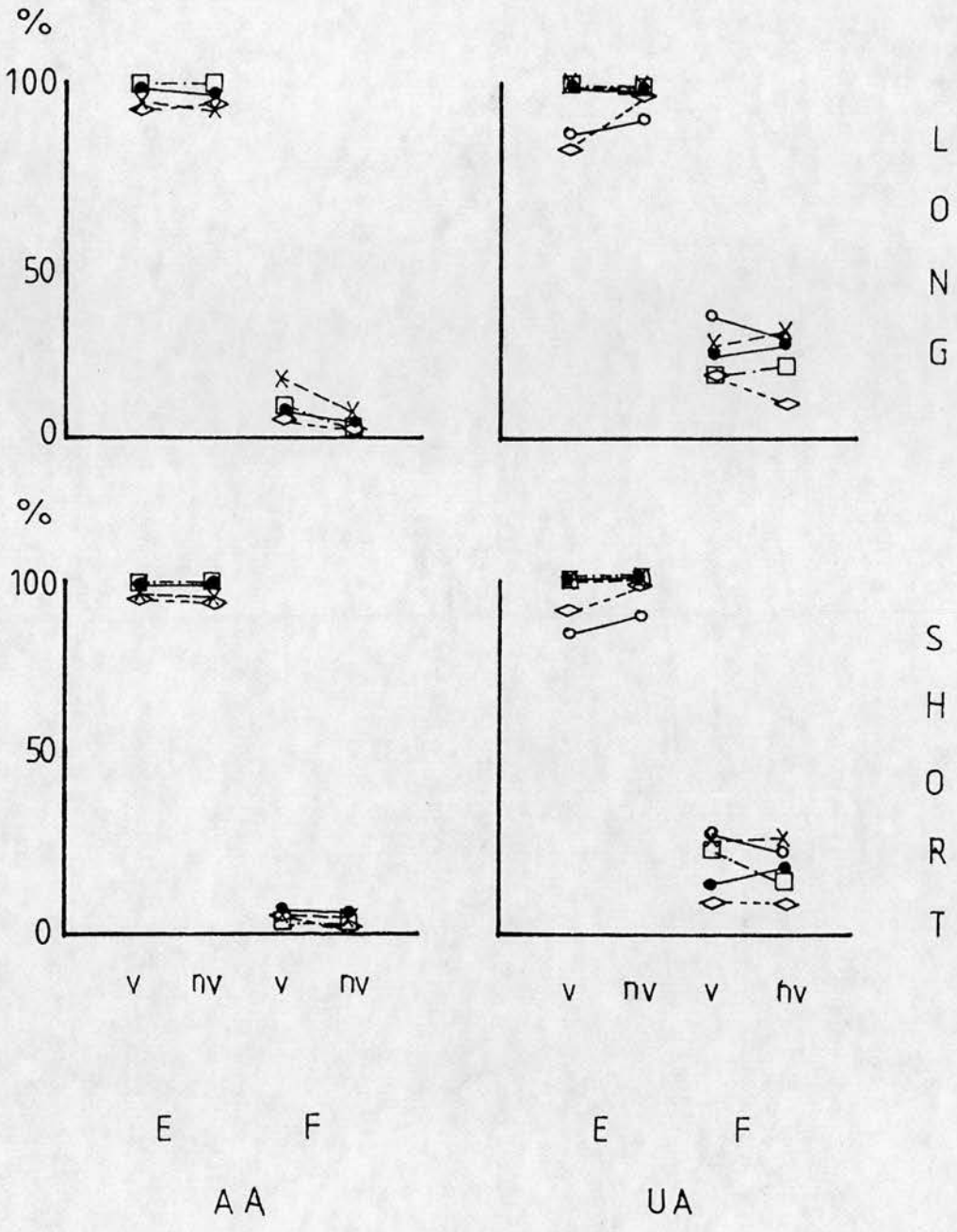



Figure 3.1.8. MEAN PERCENTAGE TRICEPS ACTIVITY

Table 3.1.13
ANOVA Results for Triceps Activity

| SOURCE OF VARIATION | MS | SUBJECTS EW | JG | WI |
|---|---|---|---|---|
| ARM | 2.376 | 40.468 | 36.063 | 11.839 |
| | ns | *** | *** | *** |
| DIR | 1530.077 | 5003.332 | 1847.055 | 1608.736 |
| | *** | *** | *** | *** |
| VIS | 0.369 | 2.761 | 0.251 | 0.028 |
| | ns | ns | ns | ns |
| DIST | 0.000 | 0.166 | 1.047 | 0.906 |
| | ns | ns | ns | ns |
| ARM X DIR | 7.836 | 40.468 | 20.453 | 10.526 |
| | ** | *** | *** | ** |
| ARM X VIS | 1.510 | 0.215 | 1.345 | 1.104 |
| | ns | ns | ns | ns |
| DIR X VIS | 6.506 | 2.761 | 0.011 | 0.001 |
| | * | ns | ns | ns |
| ARM X DIST | 0.277 | 0.195 | 0.055 | 1.217 |
| | ns | ns | ns | ns |
| DIR X DIST | 1.787 | 0.166 | 1.939 | 1.217 |
| | ns | ns | ns | ns |
| VIS X DIST | 0.007 | 0.330 | 0.000 | 0.001 |
| | ns | ns | ns | ns |
| ARM X DIR X VIS | 1.348 | 0.215 | 0.584 | 0.730 |
| | ns | ns | ns | ns |
| ARM X DIR X DIST | 1.042 | 0.195 | 0.364 | 0.823 |
| | ns | ns | ns | ns |
| ARM X VIS X DIST | 0.002 | 2.028 | 0.164 | 0.013 |
| | ns | ns | ns | ns |
| DIR X VIS X DIST | 0.714 | 0.330 | 0.193 | 0.027 |
| | ns | ns | ns | ns |
| ARM X DIR X VIS X DIST | 0.847 | 2.028 | 0.716 | 0.007 |
| | ns | ns | ns | ns |

| | |
|---|---|
| ns | non-significant |
| * | $p < 0.05$ |
| ** | $p < 0.01$ |
| *** | $p < 0.001$ |

during extension.

All the hemiparetic subjects also show a main effect of Dir for the same reason as AL. However all of them also have significant Arm x Dir interactions and all but MS show main effects of Arm. For each, post-hoc analysis reveals this to indicate significantly more triceps activity during UA, as opposed to AA flexion. Finally, MS has a significant Dir x Vis interaction, brought about by greater activity in the NV condition for the UA.

Biceps - Table 3.1.14 contains the means and standard deviations of biceps activity. The means are plotted in Figure 3.1.9 with the ANOVA results presented in Table 3.1.15. Analysis of AL's data produced a main effect of Dir due to significantly higher biceps activity during flexion movements. There is also a lesser, but yet significant main effect of vision which indicates a lowering of activity under NV conditions. Although none of the relevant interactions are significant, inspection of Figure 3.1.9 shows the principal effect of this to be on extension movements to the short distance target.

A similar pattern of main effect of Dir is present in all the hemiparetic data and for the same reason. In all of the patients biceps activity is greater in the AA and is significant as a main effect of Arm in all but JG. As Figure 3.1.9 illustrates the cause of this trend is due to an increase in activity during AA extension when

Table 3.1.14
Mean (S.D.) Percentage Biceps Activity

| CONDITIONS | | | SUBJECTS EW | JG | MS | WI | AL |
|---|---|---|---|---|---|---|---|
| | E | V L | 6.45 | 11.36 | 14.16 | 12.70 | 26.10 |
| | | | (15.27) | (18.70) | (13.71) | (21.98) | (16.84) |
| | | S | 6.72 | 20.24 | 12.53 | 5.21 | 38.70 |
| | | | (10.93) | (21.51) | (27.80) | (11.61) | (23.05) |
| | NV | L | 5.50 | 12.23 | 21.22 | 12.76 | 22.60 |
| | | | (7.25) | (14.80) | (14.09) | (21.92) | (20.97) |
| UA | | S | 2.56 | 14.84 | 24.86 | 10.95 | 13.40 |
| | | | (6.16) | (19.33) | (17.73) | (17.77) | (18.67) |
| | F | V L | 95.99 | 98.60 | 89.33 | 97.49 | 91.70 |
| | | | (9.10) | (4.43) | (31.36) | (7.94) | (10.46) |
| | | S | 98.87 | 100.00 | 90.57 | 94.80 | 90.30 |
| | | | (3.57) | (0.00) | (10.02) | (13.15) | (7.22) |
| | NV | L | 96.19 | 97.35 | 87.33 | 100.00 | 88.40 |
| | | | (6.88) | (4.44) | (13.00) | (0.00) | (10.77) |
| | | S | 96.07 | 94.89 | 93.98 | 100.00 | 91.10 |
| | | | (8.57) | (11.23) | (6.98) | (0.00) | (10.84) |
| | E | V L | 58.19 | 23.82 | 41.61 | 35.36 | |
| | | | (11.87) | (22.98) | (22.61) | (21.36) | |
| | | S | 63.09 | 2.73 | 40.30 | 36.26 | |
| | | | (22.43) | (8.63) | (12.47) | (19.40) | |
| | NV | L | 80.66 | 40.14 | 66.25 | 67.18 | |
| | | | (9.88) | (23.39) | (13.21) | (11.70) | |
| AA | | S | 81.53 | 38.12 | 66.20 | 71.21 | |
| | | | (8.86) | (34.28) | (16.50) | (15.47) | |
| | F | V L | 100.00 | 93.05 | 100.00 | 100.00 | |
| | | | (0.00) | (9.57) | (0.00) | (0.00) | |
| | | S | 99.04 | 93.03 | 100.00 | 100.00 | |
| | | | (2.04) | (10.88) | (0.00) | (0.00) | |
| | NV | L | 100.00 | 96.22 | 100.00 | 100.00 | |
| | | | (0.00) | (4.93) | (0.00) | (0.00) | |
| | | S | 91.07 | 94.97 | 100.00 | 100.00 | |
| | | | (13.67) | (8.10) | (0.00) | (0.00) | |

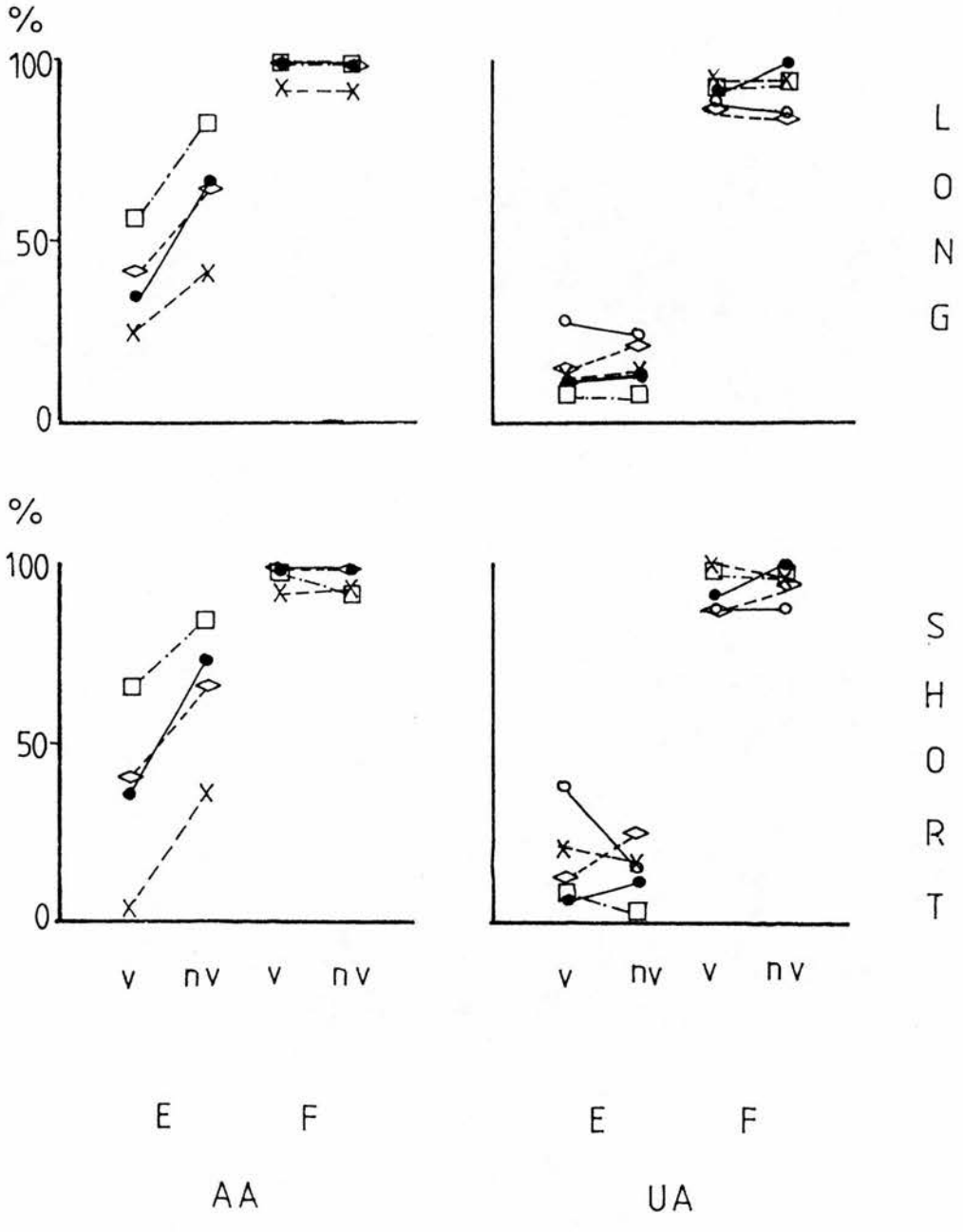



Figure 3.1.9 MEAN PERCENTAGE BICEPS ACTIVITY

Table 3.1.15
ANOVA Results for Biceps Activity

| SOURCE OF VARIATION | MS | SUBJECTS EW | JG | WI |
|---|---|---|---|---|
| ARM | 83.665 *** | 421.083 *** | 2.499 ns | 108.475 *** |
| DIR | 877.893 *** | 1336.537 *** | 861.345 *** | 1022.489 *** |
| VIS | 13.087 *** | 3.810 ns | 4.970 * | 22.544 *** |
| DIST | 0.188 ns | 0.097 ns | 0.459 ns | 0.174 ns |
| ARM X DIR | 27.171 *** | 402.308 *** | 8.400 ** | 90.309 *** |
| ARM X VIS | 2.274 ns | 9.891 ** | 10.802 *** | 9.922 ** |
| DIR X VIS | 12.071 *** | 12.871 *** | 5.526 * | 14.715 *** |
| ARM X DIST | 0.326 ns | 0.106 ns | 2.855 ns | 1.001 ns |
| DIR X DIST | 0.135 ns | 0.626 ns | 0.203 ns | 0.010 ns |
| VIS X DIST | 0.336 ns | 1.985 ns | 2.855 ns | 1.001 ns |
| ARM X DIR X VIS | 2.726 ns | 15.808 *** | 4.722 * | 16.499 *** |
| ARM X DIR X DIST | 0.053 ns | 2.662 ns | 2.787 ns | 0.466 ns |
| ARM X VIS X DIST | 0.228 ns | 0.201 ns | 1.843 ns | 0.096 ns |
| DIR X VIS X DIST | 0.003 ns | 0.083 ns | 0.754 ns | 0.131 ns |
| ARM X DIR X VIS X DIST | 0.005 ns | 0.103 ns | 1.215 ns | 0.000 ns |

| | |
|---|---|
| ns | non-significant |
| * | $p < 0.05$ |
| ** | $p < 0.01$ |
| *** | $p < 0.001$ |

vision of the arm is occluded. Post-hoc analysis of the broadly significant main effects of Vis and Arm x Dir, Arm x Vis, Dir x Vis and Arm x Dir x Vis interactions, confirm this interpretation.

Summary of Results

The movements produced by the normal subject (AL) fit with the results on similar measures from other studies. Performance is highly accurate in all conditions, with with loss of vision raising the standard deviation. This probably reflects greater uncertainty in: a) knowledge of starting position; this has to be inferred from the position of the starting strip and b) the hand/target positional relation in the final homing-in phase. Her movements are fast, showing the typical Fitts' Law effect of a reduction in velocity when movement is halved, with initiation times around the normal 200 msecs. However points (a) and (b) are also likely to account for the reduction in velocity found when vision is withdrawn and point (a) may be the cause of the raising of IT in these conditions as well. Movement is also smooth with a large percentage of the trajectory contained within one submovement. Also the mean submovement distance halves when the target distance halves. The EMG data shows the normal pattern for reasonably fast movements of some antagonist activity with predominant agonist activity.

When the patients' results are examined a fairly consistent pattern emerges. Whilst the unaffected arm is only marginally less accurate than normal, it does differ on two scores:-

1) IT is higher than normal.

2) The mean velocity is lower.

Inter-individual differences in unaffected arm performance are high, making any other comment on it uncertain.

The results for the affected arm differ quite markedly, but the difference is primarily on extension and in particular when there is no sight of performance. Under these conditions, the affected arm is less accurate. This is mostly due to the undershoot of extension. In contrast to this, there is some evidence that flexion of the affected arm without vision overshoots the mean position attained with full vision. Affected arm movements are generally slower. However,again, this is primarily due to the weakening of extension when vision is occluded. The same combination of extension and NV is responsible for the low mean submovement distance found for the affected arm. These movements take so long, is because they are jerky.

As with the unaffected arm, the manipulation of conditions does not consistently affect the higher than normal IT observed. However the EMG patterns across the arms are quite different. Whilst the unaffected arm across DIR exhibits a pattern of predominantly agonist activity with little antagonist activity, there are clear effects of DIR on affected arm performance. In flexion

the amount of antagonist triceps activity is lower than in the unaffected arm, but in extension the activity of the antagonist biceps is greater than in the other arm. The affected arm therefore shows an asymmetry in antagonist activity across flexion and extension.

## Discussion

This experiment was designed to test and expand upon the major conclusion of Chapter II: that continuous visual guidance of affected arm extension appears necessary for accurate performance. The constant error results of this experiment confirm that hypothesis, that with the kinematic analysis suggesting how vision might be functioning during movement execution.

In normal arm movements and movements of the unaffected arm, a large proportion of the normal and unaffected trajectory is achieved within one submovement, suggesting any later submovements to be indicative of small final corrections. Thus the control processes underlying these movements largely use visual information about the task in a feed-forward manner to pre-program the movement. Carlton (1981) has demonstrated that during movement execution vision only becomes important in the latter half of a trajectory. He points out that in normals, information about the position of the arm early in the pre-programmed phase, gives little indication of any fine correction which may be necessary toward the completion of the action.

The data on affected arm performance suggests quite a different control process. The movements are slow and

jerky. The extension movements of the affected arm extension are not composed of one major submovement with subsequent fine correction, but rather are fractionated into a series of submovements, each encompassing only a small percentage of the total target distance. This supports the hypothesis raised in Chapter II, that extension movements of the affected arm are continuously controlled by vision, rather than pre-programmed. This point is reinforced by the failure to find significant differences on extension across the two target distances. The consistent finding in normals, ie pre-programming of a higher velocity, for greater target distance, does not occur.

An important aspect of the results is the effect of visual monitoring on both the mean velocity and the mean submovement distance, of affected arm extension. Under visual guidance the movement is faster and can be sustained over a greater distance. This latter point raises the question as to whether the abnormally high number of submovements found, is truly representative of a correcting process, in the sense usually discussed in the literature. For example, in one of the papers quoted earlier in this Chapter (von Hofsten,1980), high numbers of submovements were found in the trajectories of reaching movements by young infants. The task for the babies consisted of reaching with a free arm to trap an attractive moving object. When the data is examined it

becomes apparent that the high number of submovements is due to zig-zag ongoing corrections aimed at keeping the infant on course for future contact with the object. The task in this experiment is clearly different. Since the movement is physically constrained to one dimension, there is no necessity for corrections of deviations from a line to the target. Therefore the only corrections necessary are if the subject does not produce enough force to carry the hand to the target. However if, as has been argued, such corrections are visually based, then why does the mean submovment distance increase under visual guidance? It seems more likely that the jerkiness in extension is due to the action being interrupted, rather than corrected. The EMG data indicates that this is probably the case.

The presence of high biceps activity during affected arm extension in general, seems paradoxical, bearing in mind that slow movements typically exhibit very little antagonist activity. Contraction of the antagonist has been found to be necessary only in fast movements when the visco-elastic forces brought about by stretch of the antagonist are insufficient to counteract the torque produced by the agonist (Marsden et al, 1983; Lestienne, 1979). Data on normal subjects indicates the CNS can, to a large extent, pre-plan for the necessity of antagonist contraction: Instructions regarding amplitude and speed of movement have been shown to affect whether or not the

antagonist fires, and if so, to what degree it is active (Marsden et al, 1983; Lestienne, 1979). An example of pre-planning comes from one of the conditions in the Marsden et al (1983) study. Subjects were instructed to produce a fast movement which they knew would be halted by a stop. In this situation, they produced movements comparable in extent and peak velocity to those where the target was visually, rather than mechanically, defined but with minimal antagonist activity. Marsden et al (1983) propose that when the subject knows that antagonist activity is required, the CNS is automatically capable of adjusting the size and time of onset necessary for accurate braking of the movement.

In the slow extension movements of the affected arm observed in this study, the presence and persistence of biceps activity, concommitant with a low mean velocity and much discontinuity, may be viewed as revealing a failure in the normally flexible control of antagonist function, leading to inappropriate coactivation. In the presence of approximately 100% triceps activity it is reasonable to propose that the discontinuity in extension probably results from interruption to movement by the hyperactive antagonist. These arguments support the hypothesis raised in Chapter II, that failure to reach the target when extending the affected arm may be due to information from the uninhibited antagonist stretch receptors leading the system to "believe" the angle at

the elbow is greater than it really is. Additionally, the early cessation of movement may be caused, in part, by the torque produced by the overactive antagonist biceps.

The most significant aspect of the results of this experiment, is the difference in affected arm extension across the two visual conditions. Under full visual guidance, extension is less jerky, faster, and accompanied by less biceps co-contraction, in addition to being more accurate. Therefore it looks as if in some way, visual information concerning the nature of performance can aid in switching off unnecessary antagonist co-contraction. This specific point is further explored in Experiment 3.3.

Another way in which visual guidance may be functioning could be by allowing the subject a more realistic appreciation of the sense of effort involved in performing extension. Stroke patients often complain that the affected arm feels heavy and tires easily (Gandevia and McCloskey, 1977). Wundt (1863) (cited in McCloskey, 1981, p. 1416), noted the patient has "a sense of more work effected than formerly, and yet the effected work is the same or even less". Verbal reports from subjects participating in this experiment indicated that this was the case. Often when performing with the affected arm occluded, they would suddenly halt the

movement and say " I must be there by now." It could be that when extending the affected arm without sight of it, the patient monitors the amount of effort inputted to the task, thus receiving a false impression of how far it has actually moved. When he can view performance however, the results of his efforts are clearly obvious and he sustains motor output until the target criterion is attained. This hypothesis, that without vision, control of the hemiparetic limb is dominated by "sense of effort", is examined in Experiment 3.2.

In contrast to extension, flexion of the affected arm does not undershoot the target under NV and if at all, tends slightly to overshoot the mean end-point postion achieved under FV. Additionally there is some evidence that velocity increases under NV rather than decreasing. Also, although more discontinuous, affected arm flexion resembles unaffected performance more than it does affected arm extension. This therefore supports the idea that flexion precedes extension in recovery.

During flexion, the triceps was less active in the affected arm than in the unaffected, despite the trend for movements of the same extent to have similar mean velocity values. However, in spite of this, and the possibility of hyperexcitability in the flexor structures, the prediction that flexion would be faster and more ballistic under NV, with an overshoot comparable

to the undershoot observed for extension, has not been upheld. Therefore although when serving as an antagonist, control of the biceps is abnormal, when functioning as an agonist, control of its activity appears relatively normal, suggesting any release from supraspinal inhibition to be a problem only for extension movements.

Intimately tied in with this dysfunction in control, is the ability of the CNS to utilise visually specified information to control muscle firing. In discussing the finding that antagonist activity varies as a function of movement amplitude and speed Marsden et al (1983) question how this might occur:

"Somehow the nervous system must be capable of computing this relation of distance to be moved and velocity of movement, so as to determine the size of the antagonist burst required to halt movement." (p. 2).

As this experiment demonstrates, the information necessary to achieve this can be visually picked up and then used to supraspinally modulate the state of the spinal apparatus.

## Experiment 3.2 - The effect of "sense of effort" on non-visually guided movements.

### Introduction

This experiment was designed to examine the hypothesis that one of the functions of vision, in aiding more accurate performance of the paretic limb, is to counteract a possibly misleading, somatosensorily based sense of effort.

Although Experiment 2.2 indicated that in the absence of vision the actions of both arms can be influenced by somatic proprioception from the affected arm, there may be an additional process responsible for the undershoot effect exhibited by the affected arm. If, following Yanagisawa et al (1976), overactive disynaptic inhibition is acting on the extensor motoneurons, then in order for them to fire, more than usual supraspinal excitation may be necessary. A dominant theory in the motor skills literature is that one of the methods by which the CNS monitors movement, is through a comparison of actual efferent discharge with intended. This is known as the efference copy (Jones, 1974) theory of movement control (see pages 39-40 for a fuller discussion). The idea is that simultaneous with corticofugal discharge, a copy of intended efference is

monitored and matched with actual efference. If more efferent drive is needed than normal to contract the extensor, and the system does not take this into account when initially deciding on the amount of efference necessary for achieving the goal then, in the absence of vision, once the decided amount of efferent signal is reached, the movement will be halted, even although it has not covered the target distance.

As in the previous experiment, the task used here is derived from the linear positioning paradigm. Tasks akin to this have been used extensively to study whether location or distance cues are encoded in short- term memory for movement (Laabs, 1973; Stelmach et al, 1975; Kelso, 1977). A general conclusion from these studies is that movement is programmed predominantly on the basis of target location rather than extent of target distance (Kelso and Holt, 1980).

The hypothesis to be tested in this experiment runs as follows: if, in the absence of vision, paying attention to a false sense of effort is responsible for the paretic arm undershooting the target, then it might be expected that varying the target distance, through altering the start position, but keeping the target position constant, could produce a similar pattern of variation in movement end-point. For example, if a target distance of 15 cm on one trial is increased to 20

cm on the next, by moving the start position 5 cm further away from the target, will the arm stop moving at a point 5 cm back from the end point of the previous trial?

## Method

### Design

The experimental task was similar to that of Experiment 3.1. However in this experiment all of the movements were only in the direction of extension and performed with the arm visually occluded. A repeated measures design was employed (2 x 4). The two factors were:-

1) Arm - Affected (AA), Unaffected (UA).

2) Distance (Dist) - Medium1 (M1), Medium2 (M2), Long (l), Short (S).

Therefore there were 8 conditions with six trials per condition.

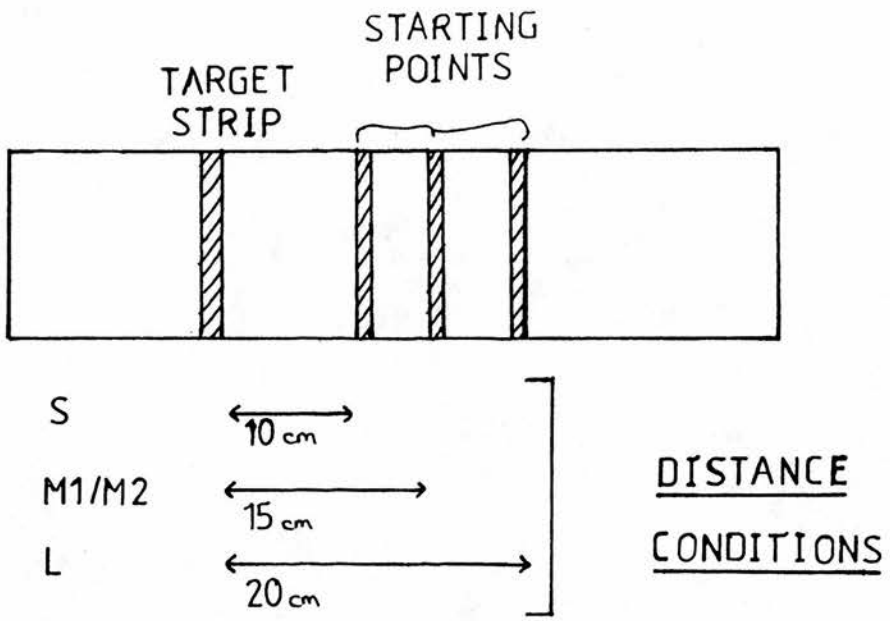
### Subjects

The subjects who participated in Experiment 3.1 also took part in this experiment.

### Apparatus

The same apparatus and experimental situation were used as in Experiment 3.1. However since the interest of this experiment lay solely with a comparison of accuracy measures, the EMG equipment was not employed. The layout of the marker strips on the perspex and cardboard surfaces was slightly different. During the experimental trials, when the occluder was in place, they were positioned on the perspex as shown in Figure 3.2.1. However on the surface of the cardboard only the target

## STRIP LAYOUT




S : SHORT = 10cm

M1/M2 : MEDIUM = 15cm

L : LONG = 20cm

FIG. 3.2.1.

strip and the 15cm start position were visible.

Procedure

As this experiment involved less conditions and trials per condition, than the previous one, it was conducted within one session. The procedure was similar to a NV condition of the previous experiment, but EMG recording was omitted and it was limited to extension movements. Also, as in the previous experiment, practice trials under FV conditions were conducted using the 15cm start position only. Following this the other two starting positions strips were placed on the perspex and the occluder and cloak installed. Instructions, as to the nature of the movement to be made, were similar to the previous experiment and emphasised that the subject must endeavour to line the marker on the cradle up under the target strip. The subject was informed that soon after commencement of trials the starting position would randomly vary among three starting positions.

Prior to the signal to move, as given by the handswitch, was a verbal "Ready" signal, plus a manual reminder of target position, followed by a variable foreperiod of up to six seconds. Criteria for the end of a trial and its acceptability were as in Experiment 3.1. On completion of a trial the cradle and arm were passively returned to the start. Six trials at the 15cm starting position (M1) were conducted. There was no

indication after these trials that the starting position was about to vary. Starting position on the next eighteen trials was determined by a random presentation of the M2, S and L conditions.

Data Analysis

The data was extracted from the tape and stored by the methods used in Experiment 3.1 Since only information on constant error was desired, recording only took place from the potentiometer channel. Constant error was obtained by calculating the difference, on each trial, between the subject's end position and the target. A two-way independent measures ANOVA, 2 x 4 (Arm x Direction) was then performed on each subject's results and post-hoc comparisons conducted using related t tests.

## Results

### Constant Error

Means and standard deviations of this measure are listed in Table 3.2.1, with the means plotted in Figure 3.2.2 and the ANOVA results presented in Table 3.2.2. The normal subject (AL) shows no significant differences and thus demonstrates no effect of variation in starting position upon movement accuracy.

All the hemiparetic subjects exhibit main effects of Arm because movements of the affected arm are less accurate and undershoot the target. Three of them (MS, JG and WI) show main effects of Dist. Post-hoc analysis, taking into account two Arm x Dist interactions being significant revealed no differences between the M1 and M2 conditions. As Figure 3.2.2 shows, there is a tendency for a greater undershoot at the 20cm distance, but this is only significantly different from the other distances for subjects MS and WI. The figure also demonstrates that in this condition subject MS produced a similar (and significant) result with the UA as well. The only instance of a significant overshoot in the S condition was from the UA of subject WI.

Table 3.2.1
Mean (s.d.) Constant Error (cm)

AFFECTED ARM

| SUBJECT | DISTANCE 15A | 15B | 20 | 10 |
|---|---|---|---|---|
| MS | -3.16 | -5.56 | -10.90 | -3.43 |
| | (2.71) | (1.79) | (0.65) | (2.50) |
| EW | -6.24 | -2.90 | -4.22 | -1.22 |
| | (1.23) | (0.96) | (1.95) | (0.92) |
| JG | -1.89 | -2.39 | -4.62 | -2.28 |
| | (0.88) | (0.81) | (0.57) | (0.60) |
| WI | -3.56 | -3.43 | -5.50 | -2.64 |
| | (0.49) | (0.64) | (0.85) | (1.07) |

UNAFFECTED ARM

| SUBJECT | DISTANCE 15A | 15B | 20 | 10 |
|---|---|---|---|---|
| MS | -1.20 | +0.33 | -7.00 | +1.58 |
| | (2.63) | (4.33) | (3.57) | (1.25) |
| EW | +1.29 | +0.62 | +0.33 | +1.30 |
| | (2.30) | (3.44) | (4.22) | (1.83) |
| JG | +0.87 | +2.59 | +2.95 | +6.25 |
| | (1.97) | (2.73) | (2.57) | (3.19) |
| WI | +1.21 | +2.10 | +1.80 | +3.51 |
| | (0.68) | (1.05) | (0.87) | (0.79) |
| AL | -0.41 | +0.98 | +0.81 | +0.82 |
| | (0.58) | (0.85) | (0.72) | (1.19) |

# UA

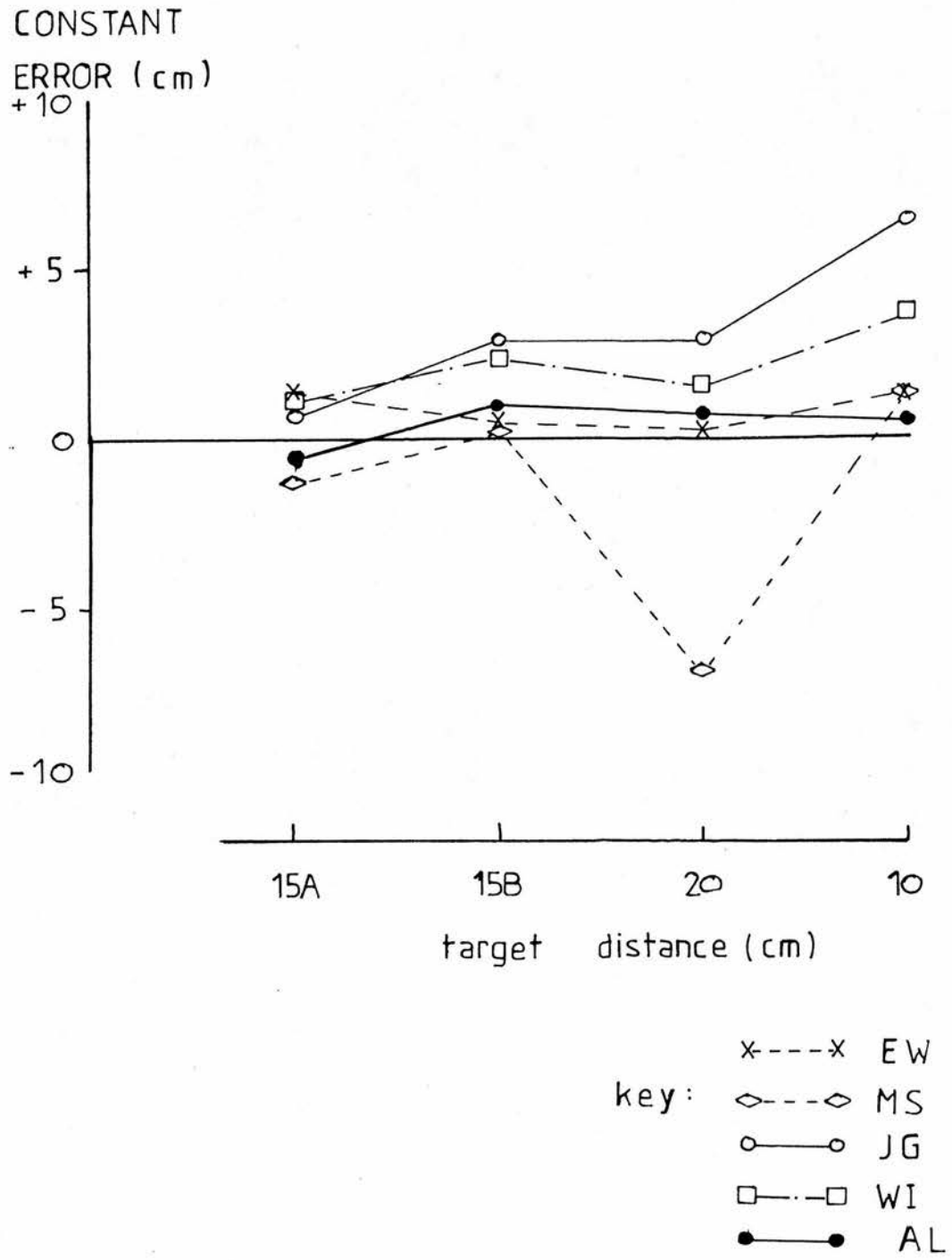



Figure 3.2.2.A

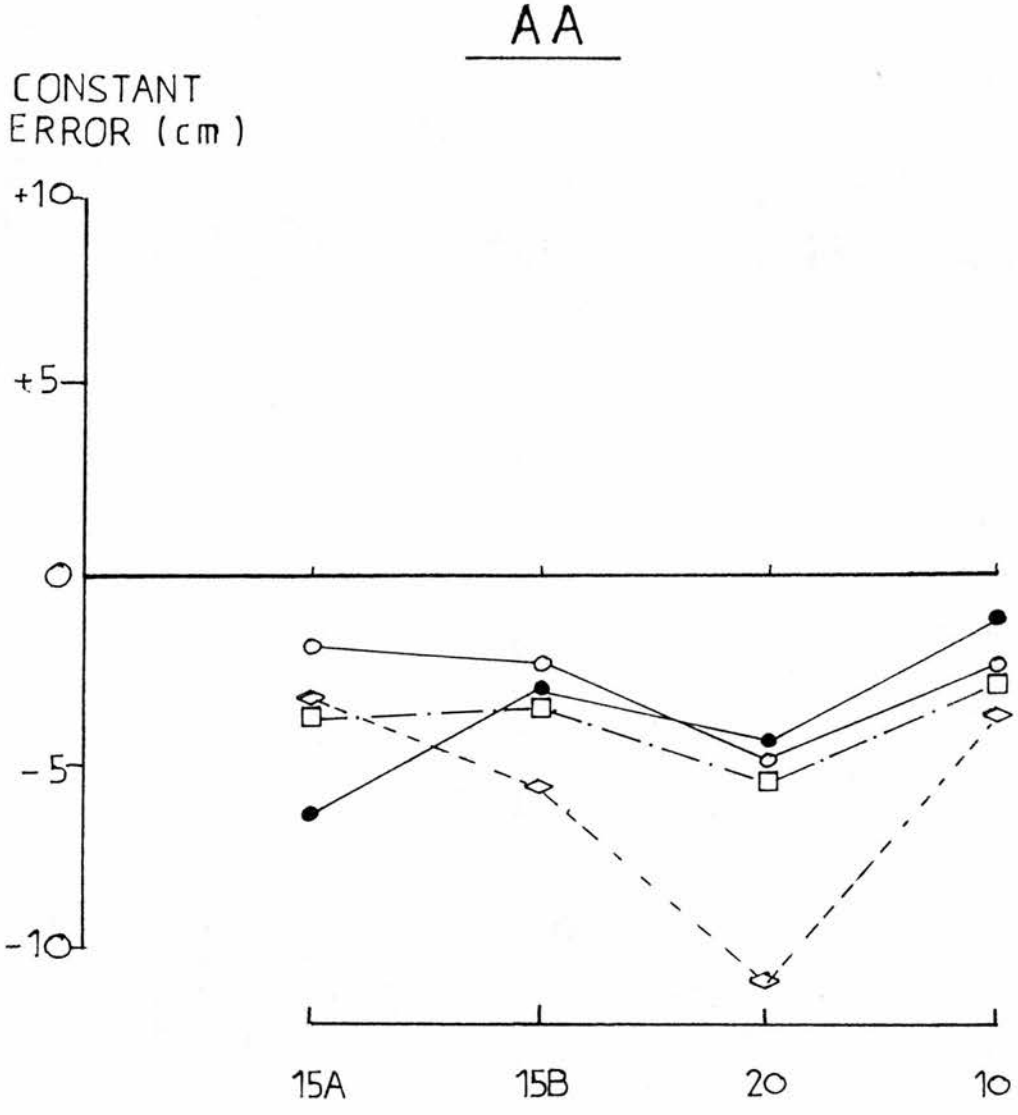



Figure 3.2.2.

Table 3.2.2
Anova Results for Constant Error

| SOURCE OF VARIATION | SUBJECTS WI | MS | EW | JG |
|---|---|---|---|---|
| ARM | 618.449 | 29.478 | 43.337 | 112.736 |
| | *** | *** | *** | *** |
| DIST | 16.285 | 21.670 | 2.526 | 5.045 |
| | *** | *** | ns | ** |
| ARM X DIST | 5.009 | 1.203 | 2.486 | 5.404 |
| | ** | ns | ns | ** |

## Discussion

AL shows the typical result found with normals; ie no effect of varying starting position upon movement accuracy (Stelmach et al, 1975). However, after the first six trials the standard deviation of accuracy increases indicating a slightly greater uncertainty in estimation of target position, or of the felt position of the arm.

As in the previous experiment, all the hemiplegic subjects undershoot the target when extending the affected arm. Although significant in only two patients, there is a consistent trend for the degree of undershoot to be greater when the distance to be moved is doubled. However on the basis of the results, it is difficult to decide whether accuracy of the affected arm is determined by target location or distance. From the low numbers of significant differences across the conditions, it would appear that the arm is being directed aimed in intended direction. However, the fluctuations in error across the conditions could indicate that this intention is confounded by a false sense of effort. Therefore it appears that to a limited extent, stroke patients may falsely equate sense of effort in moving the paretic limb, with actual work done.

The results for the unaffected arm, although generally more accurate, show a similar pattern of difference between long and short distances across all the subjects. This makes uncertain any conclusion concerning the affected arm alone. What the manipulations of this experiment may have revealed is a general inflexibility in the CNS to adapt to altered circumstances in the absence of vision following stroke.

## Experiment 3.3 - Reversal of movement.

### Introduction

De Souza et al (1980) have reported that a major deficit in movement control following stroke is the inability to make a smooth transition from one muscle synergy to another - a feature of control necessary to the patterning and sequencing of movement.

In Experiment 3.1 the hypothesis was raised that a potentially important function of vision in controlling affected arm movement, is in the provision of information for the supraspinal modulation of the reciprocal timing of the agonist/antagonist linkage. If, as was argued for affected arm extension, visual monitoring of performance can aid in switching off inappropriate biceps contraction, then this should be amenable to examination by studying how quickly the arm can be reversed from a movement into flexion, to one of extension. The hypothesis would predict that withdrawal of vision would increase the time taken to make a reversal. Given the case that there may be a bias in the direction of flexion, the opposite reversal, from extension into flexion, should be faster.

## Method

### Design

In this experiment subjects were required to reverse a movement as quickly as possible. A 2 x 2 x 2 design was employed with each subject taking part in each condition. The three factors were:-

1) Arm - Affected (AA), Unaffected (UA)

2) Direction (Dir) - Extension, Flexion

3) Vision (Vis) - Full Vision (FV), No Vision (NV)

This yielded a total of eight conditions with ten trials per condition. Presentation of the conditions was randomly ordered.

### Subjects

The subjects who took part in Experiment 3.1 also participated in this experiment.

### Apparatus

The previously described track and cradle assembly was used with output from the tachogenerator only recorded on the FM tape recorder. Some minor modifications were made to the assembly as illustrated in Figure 3.3.1. These permitted the cradle being to be attached, through quick release, to either end of the track, by a length of string and a spring.

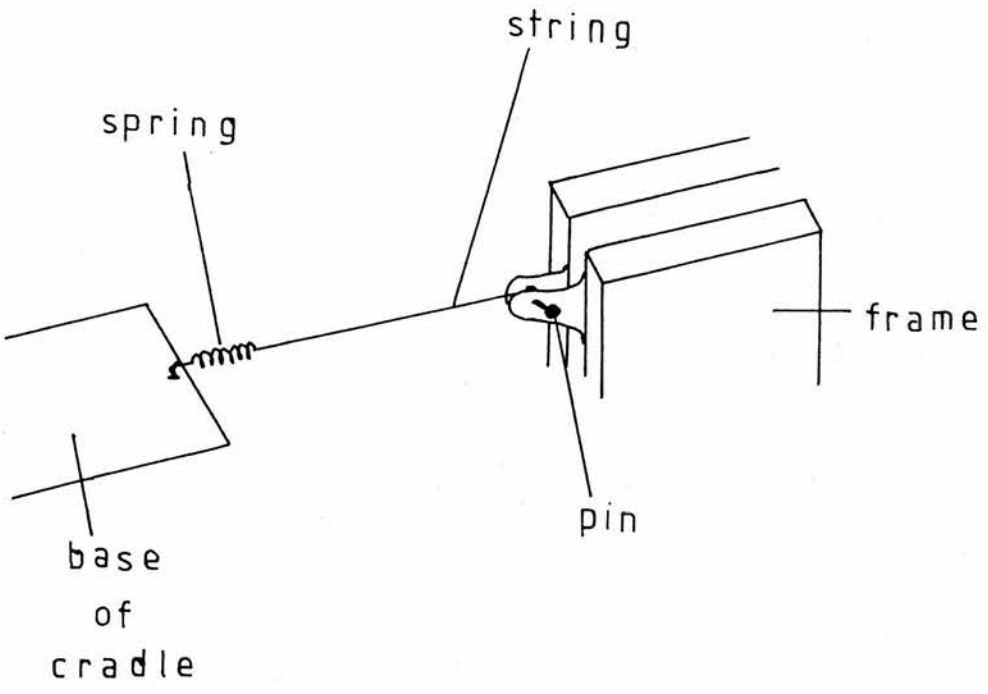



Figure 3.3.1. Connection of cradle to frame.

Procedure

For the same reasons as in Experiment 3.1, this experiment was spread over several sessions. At the initial session the subject was tested to ascertain how far he could move the cradle (Point 2) from a point on the track where the string was just taut (Point 1) against the resistance of the spring. This was done for both flexion and extension. The distances were noted and were subsequently used in all flexion or extension conditions including UA conditions.

Points 1 and 2 were visually defined by the strips of cardboard previously used as targets, either on the perspex in FV conditions, or on the occluder in NV conditions. A trial consisted of the subject moving from point 1 to point 2 and holding the cradle stationary there. In NV conditions, alignment of the pointer on the cradle was guided by the experimenter. There was then a variable foreperiod of up to eight seconds, after which time the experimenter swiftly removed the rod from the brackets on the frame, thus releasing the loop of string. This immediately caused the subject's arm to move briskly in the direction to which he had been applying resistance against the spring. The subject's task was then to halt that movement and reverse it as soon as possible. It was emphasised that halting should not be interpreted as unnecessarily pausing, but rather that halting and reversing should be attempted as one operation.

Data Analysis

The tachogenerator recordings were analysed by the procedures described in Experiment 3.1. In Figure 3.3.2 two typical records are presented. They are diagrammed in Figure 3.3.3 to illustrate the points of information extracted. Point A is the start of the movement after release of the spring. Point B is that movement's peak velocity and is therefore the commencement of deceleration. The return to zero velocity is marked by point C. In trials where halting and reversing were conducted as one operation, point C also signalled the commencement of the movement reversal. However when they were not, and there was a pause, point D marked that event.

From points A, B, C and if present D, time intervals 1,2 and optionally 3 were calculated.

# SPECIFICATION OF TIME INTERVALS

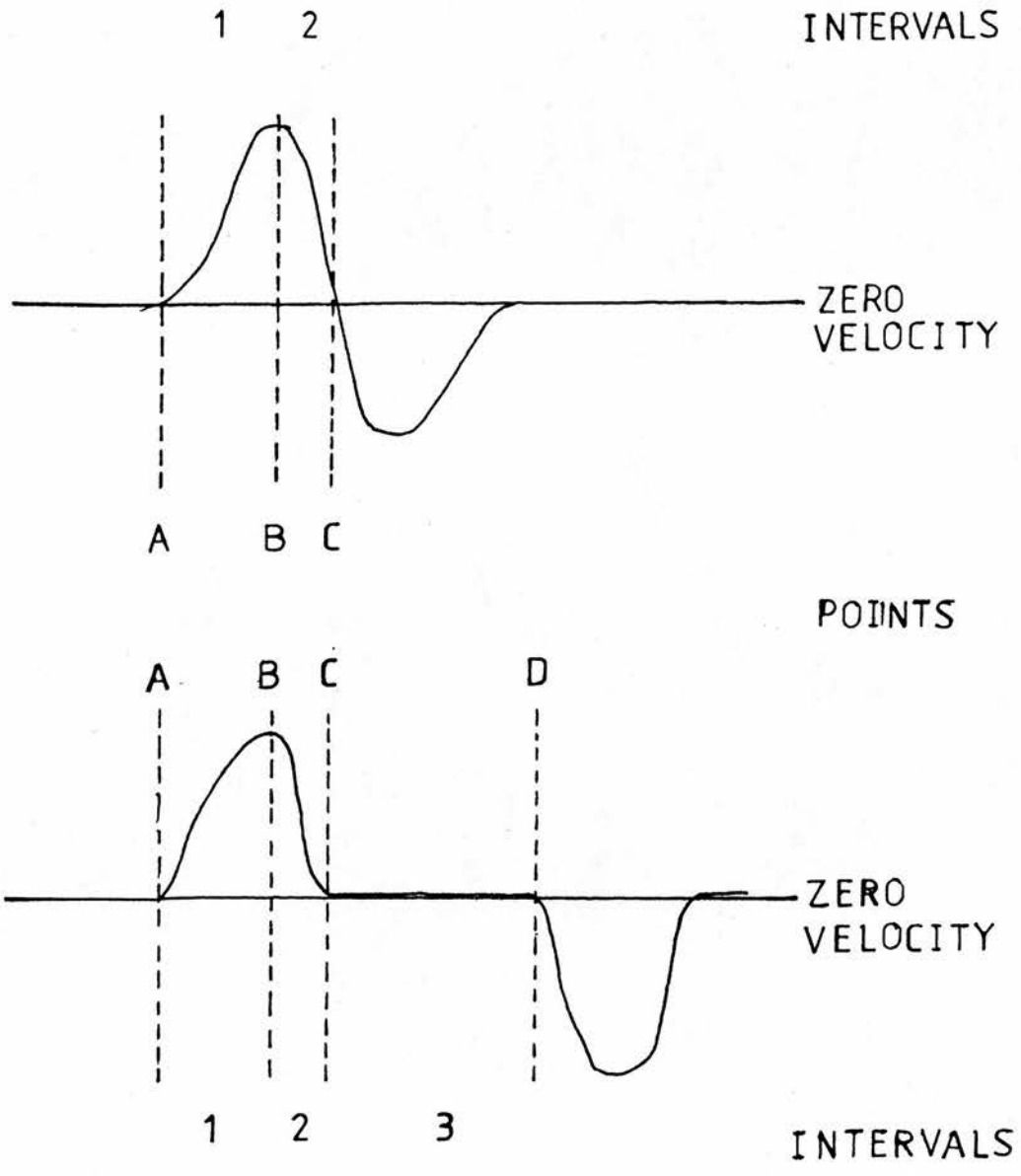
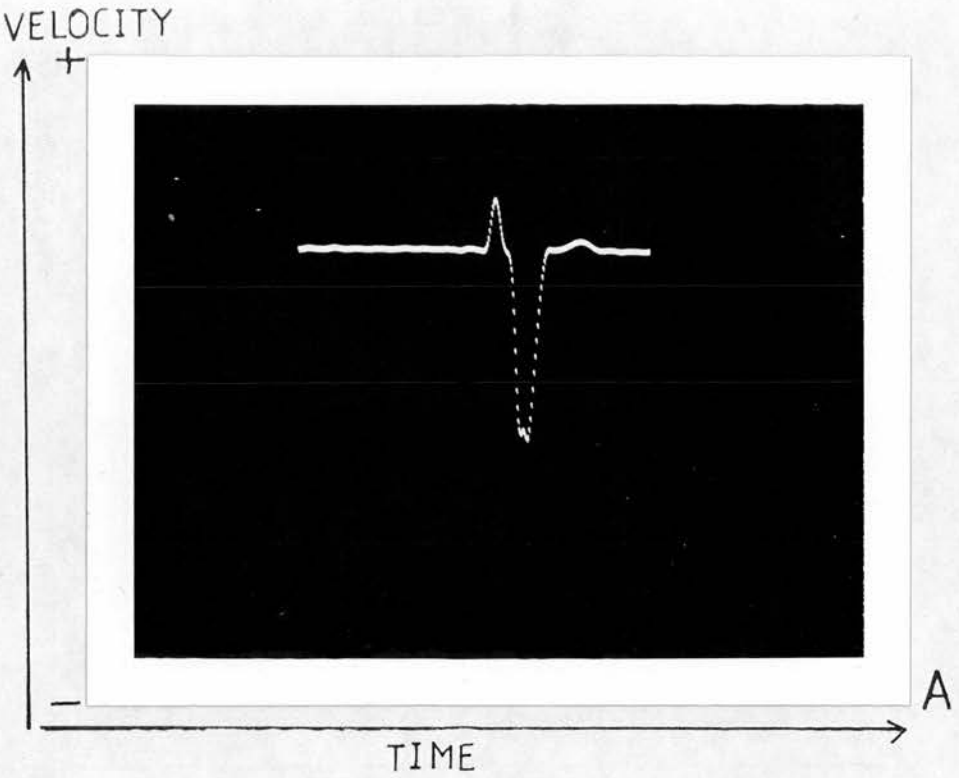



Figure 3.3.3.



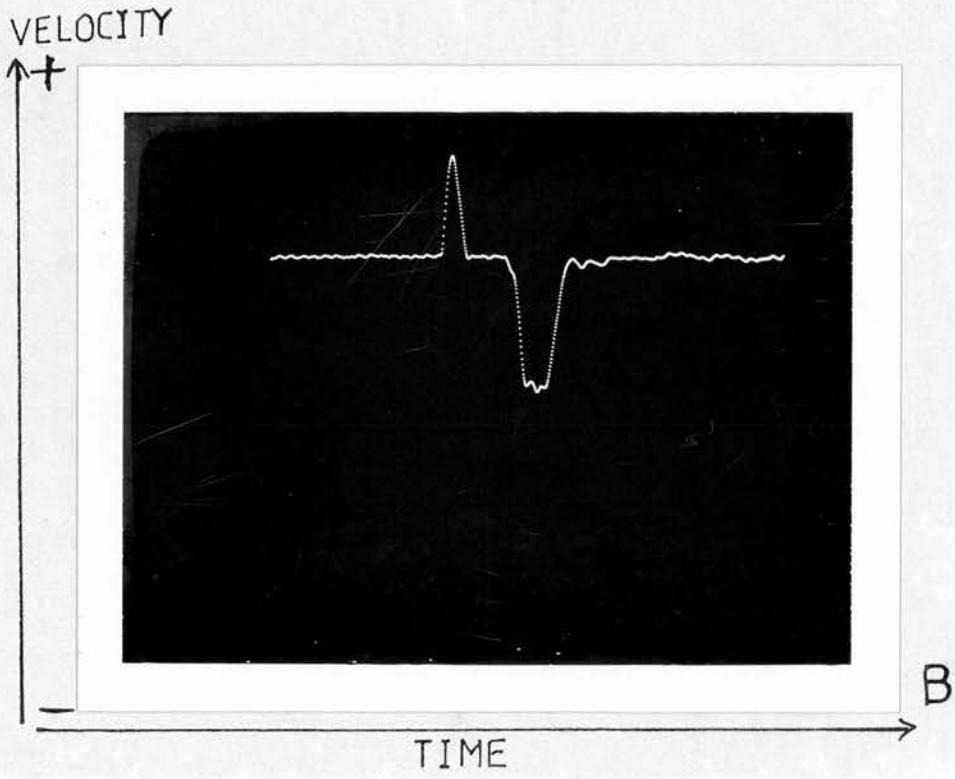




Figure 3.3.2 - Velocity profiles of movement reversals from flexion (+) into extension (-).
A : The normal arm
B : An affected arm (WI)

Results

The means and standard deviations of the time intervals are presented in Table 3.3.1 with the means diagrammed in Figure 3.3.4.

Two analyses were performed on the data:-

1) On interval 1 - The time to commence deceleration.

2) On the total time taken to commence reversal.

For each subject three-way, Arm x Dir x Vis (2 x 2 x 2), independent measures ANOVAS were carried out on these measures.

Time to commence deceleration - The ANOVA results are detailed in Table 3.3.2. Post-hoc analysis revealed the main effect of Arm obtained in all three patients to be due to the AA taking longer to commence deceleration. This was the only consistent result obtained from analysis of the main effects and interactions.

The significant Arm x Dir interactions found for EW and MS were contradictory. With MS, the difference between the arms only pertains to reversal into extension. Whereas EW only differs on reversals into flexion. Main effects of Dir were found with both WI and EW. WI commenced deceleration faster when going into extension, whilst the opposite was found for EW. In addition these two subjects show significant A x V

Table 3.3.1
Mean (S.D.) Interval Times (msec)

CONDITIONS: UA/V/EXTN

| SUBJECT | INTERVAL 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| MS | 82 | 232 | 56 | 370 |
| | (6) | (77) | (63) | (73) |
| EW | 64 | 204 | 0 | 268 |
| | (11) | (53) | (0) | (49) |
| WI | 159 | 205 | 239 | 603 |
| | (13) | (114) | (108) | (282) |
| AL | 63 | 92 | 0 | 145 |
| | (8) | (33) | (0) | (29) |

CONDITIONS: UA/NV/EXTN

| SUBJECT | INTERVAL 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| MS | 75 | 194 | 38 | 307 |
| | (8) | (63) | (42) | (42) |
| EW | 66 | 181 | 0 | 247 |
| | (5) | (20) | (0) | (20) |
| WI | 133 | 206 | 130 | 439 |
| | (8) | (101) | (264) | (136) |
| AL | 67 | 72 | 0 | 139 |
| | (5) | (29) | (0) | (30) |

CONDITIONS: UA/FLEXN/V

| SUBJECT | INTERVAL 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| MS | 113 | 224 | 45 | 382 |
| | (9) | (41) | (60) | (54) |
| EW | 100 | 106 | 0 | 207 |
| | (12) | (13) | (0) | (20) |
| WI | 135 | 171 | 89 | 405 |
| | (10) | (15) | (71) | (81) |
| AL | 97 | 93 | 0 | 190 |
| | (11) | (33) | (0) | (17) |

CONDITIONS: UA/FLEXN/NV

| SUBJECT | INTERVAL 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| MS | 118 | 186 | 154 | 458 |
| | (37) | (31) | (59) | (74) |
| EW | 95 | 124 | 0 | 219 |
| | (5) | (17) | (0) | (19) |
| WI | 124 | 156 | 211 | 491 |
| | (11) | (37) | (132) | (154) |
| AL | 90 | 101 | 0 | 191 |
| | (12) | (14) | (0) | (16) |

CONDITIONS: AA/EXTN/V

| SUBJECT | INTERVAL 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| MS | 169 | 270 | 277 | 706 |
| | (44) | (73) | (75) | (50) |
| EW | 124 | 216 | 518 | 858 |
| | (30) | (100) | (287) | (308) |
| WI | 158 | 268 | 672 | 1098 |
| | (33) | (120) | (372) | (346) |

CONDITIONS: AA/EXTN/NV

| SUBJECT | INTERVAL 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| MS | 163 | 281 | 458 | 912 |
| | (20) | (36) | (126) | (143) |
| EW | 103 | 219 | 933 | 1254 |
| | (50) | (135) | (437) | (347) |
| WI | 183 | 229 | 1188 | 1600 |
| | (12) | (70) | (222) | (439) |

CONDITIONS: AA/FLEXN/V

| SUBJECT | INTERVAL 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| MS | 129 | 231 | 192 | 552 |
| | (9) | (26) | (160) | (163) |
| EW | 111 | 152 | 348 | 611 |
| | (23) | (46) | (209) | (138) |
| WI | 131 | 210 | 487 | 828 |
| | (9) | (64) | (139) | (222) |

CONDITIONS: AA/FLEXN/NV

| SUBJECT | INTERVAL 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| MS | 123 | 190 | 194 | 507 |
| | (11) | (16) | (204) | (187) |
| EW | 101 | 170 | 200 | 471 |
| | (37) | (43) | (??) | (243) |
| WI | 134 | 318 | 648 | 1100 |
| | (12) | (114) | (227) | (104) |

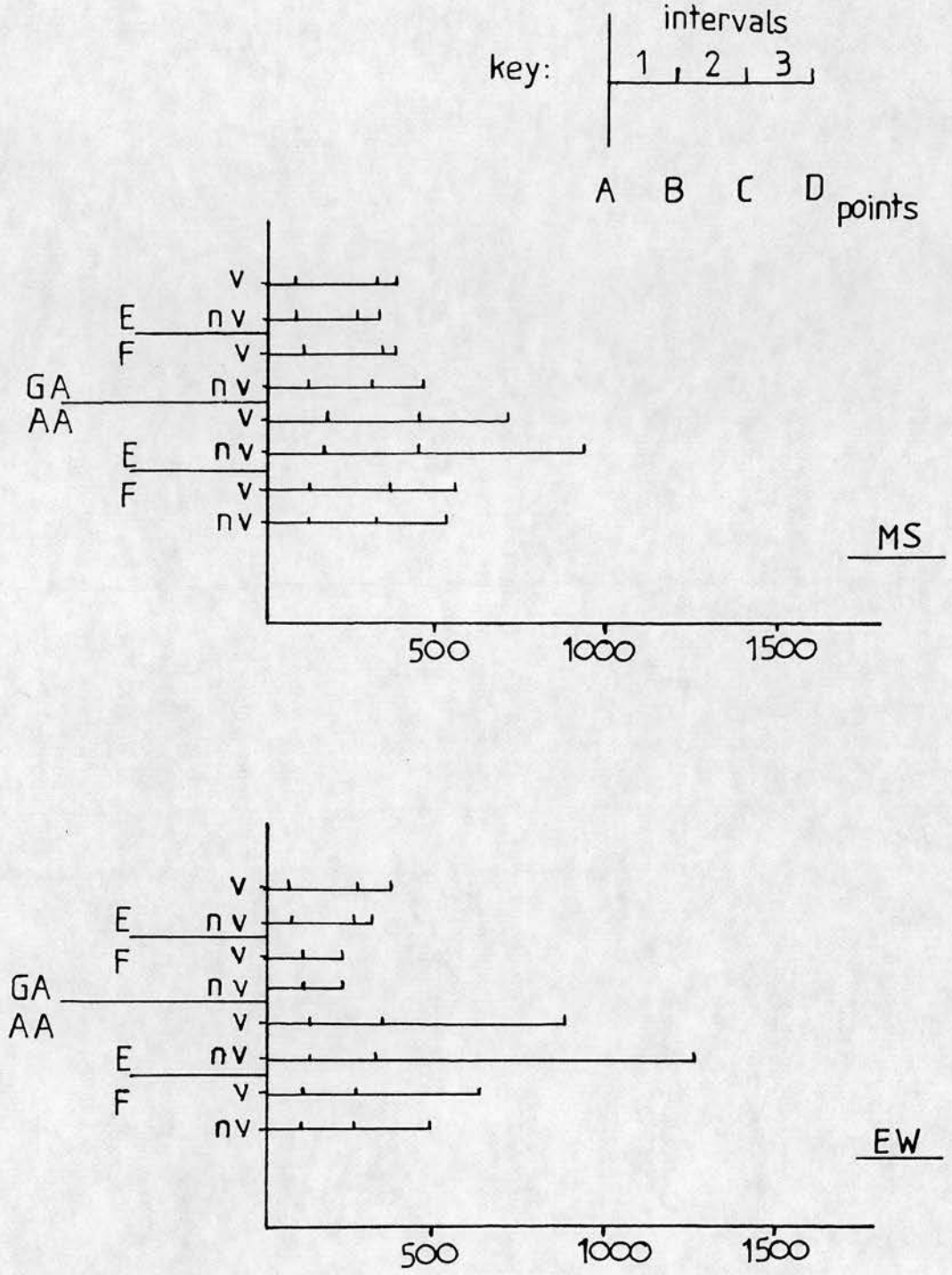



Figure 3.3.3.A. Interval Breakdown of Mean Results.

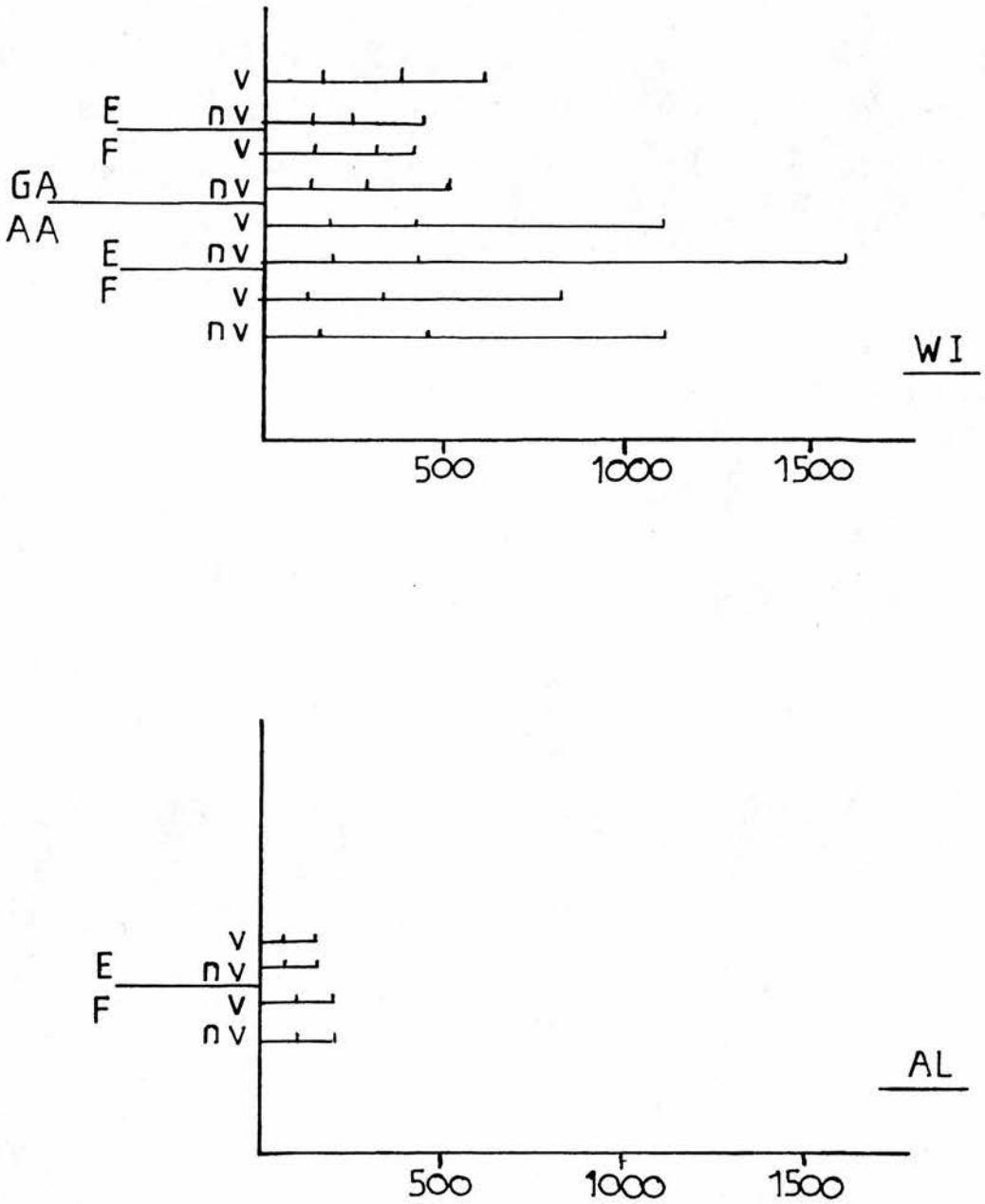



Figure 3.3.3.A. Interval Breakdown of Mean Results.

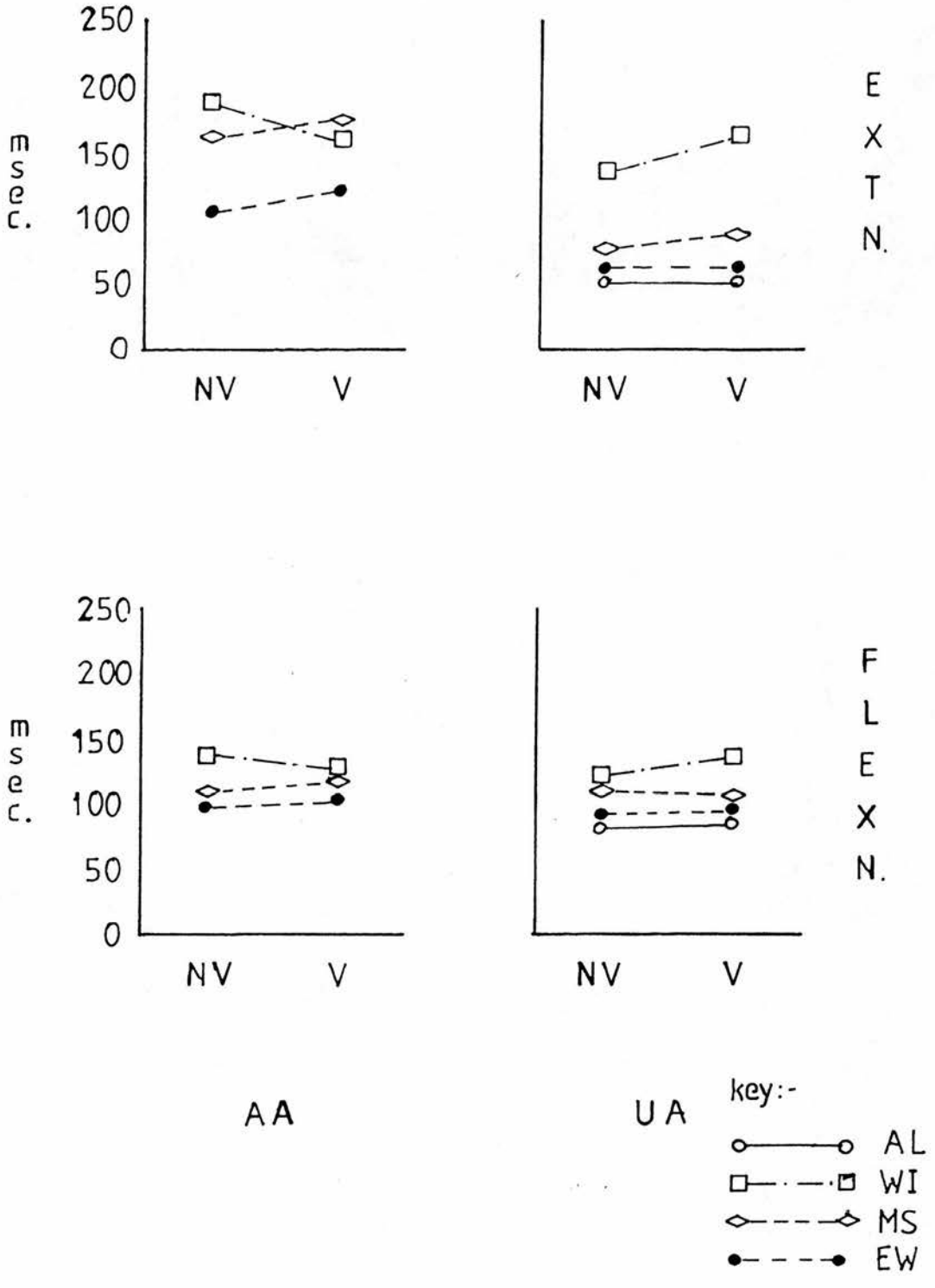



Figure 3.3.3.B. Mean Time to Commence Deceleration.

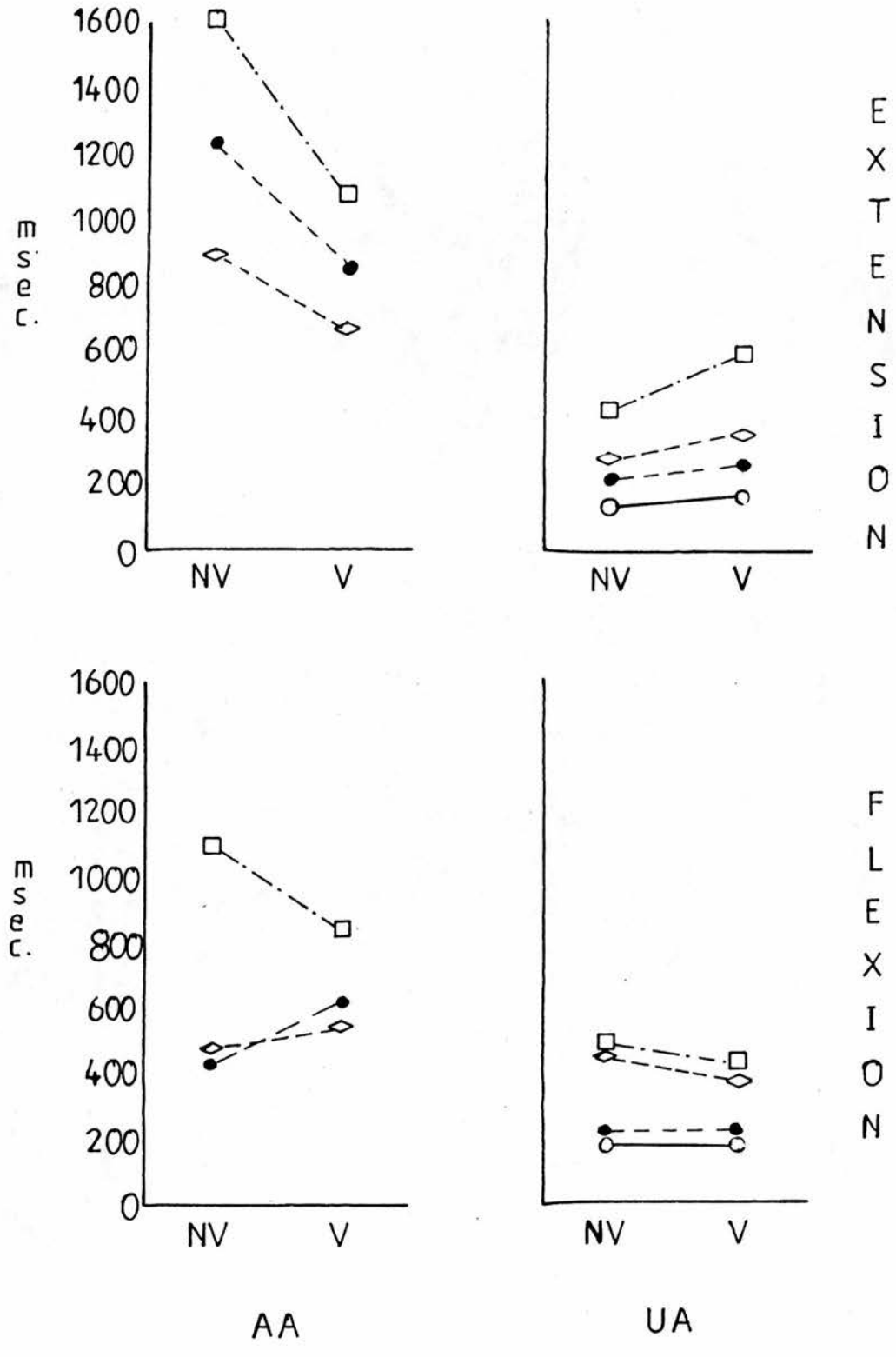



Figure 3.3.3.C. Mean Time to Commence Reversal.

Table 3.3.2
Anova Results for Commencement of Deceleration

| SOURCE OF VARIATION | MS | SUBJECTS WI | EW |
|---|---|---|---|
| ARM | 92.494 *** | 5.247 * | 73.286 *** |
| DIR | 0.087 ns | 20.607 *** | 14.096 *** |
| VIS | 0.472 ns | 0.140 ns | 6.519 * |
| ARM X DIR | 57.101 *** | 3.207 ns | 36.090 *** |
| ARM X VIS | 0.241 ns | 7.328 * | 4.421 * |
| DIR X VIS | 0.347 ns | 0.085 ns | 0.090 ns |
| ARM X DIR X VIS | 0.347 ns | 2.374 ns | 1.827 ns |

Table 3.3.3
Anova Results for Commencement of Reversal

| SOURCE OF VARIATION | MS | SUBJECTS WI | EW |
|---|---|---|---|
| ARM | 134.336 *** | 135.142 *** | 171.255 *** |
| DIR | 15.656 *** | 33.337 *** | 19.887 *** |
| VIS | 3.023 ns | 1.624 ns | 11.482 ** |
| ARM X DIR | 52.042 *** | 23.575 *** | 9.229 ** |
| ARM X VIS | 2.187 ns | 1.870 ns | 17.205 *** |
| DIR X VIS | 1.252 ns | 6.736 * | 0.009 ns |
| ARM X DIR X VIS | 15.185 *** | 8.260 ** | 5.461 * |

interactions. Again, as Figure 3.3.4 shows, the results were contradictory with respect to which arm was affected by withdrawal of vision.

The normal subject demonstrates one significant result. The main effect of Direction indicates time to commence movements into extension are shorter than into flexion.

Time to commence movement reversal - In contrast to the data on interval 1, there is a consistent pattern of results on this measure across all the hemiparetic subjects. The main effects of Arm and Dir (Table 3.3.3), indicate that all three subjects are slower with the affected arm and slower when moving from flexion into extension. These results are additive as evidenced by the significant Arm x Dir interactions. In addition the presence of significant three-way interactions qualify this finding by showing performance to be at its slowest under NV conditions. In all patients sight of the arm significantly improves upon this.

As for interval 1, AL shows only a main effect of Dir which indicates time to reverse into extension is faster than for flexion.

Discussion

An underlying assumption in discussing these results, is that the time taken to commence deceleration roughly corresponds to the time taken to start switching off the agonist, whilst the overall time to commence reversal approximately reflects the time taken for the initial antagonist to change roles and become the agonist.

The fast overall time found for the normal subject (AL) is as might be expected well within the range of a normal reaction time and illustrates the normal ability to smoothly switch from one synergy to another without pause, for example as in running. However there are two somewhat puzzling aspects to this subject's results. Firstly, contrary to other studies of reaction time to proprioceptive or visual cues (Jordan,1972; Klein and Posner, 1974), AL's results do not show significantly faster reversal when vision is withdrawn. This may have been because of the exceptionally high amount of kinesthetic information present in this experiment. Secondly, significantly, but inexplicably for both intervals, release into flexion is reversed faster.

As was predicted, the affected arm took longer to reverse from flexion into extension, than from extension into flexion. Figure 3.3.4 shows this is largely due to a long pause between halting flexion and initiating

extension. This could be taken to indicate a delay between turning off the biceps and firing the triceps. However in the presence of a strong effect of vision reducing this interval, this interpretation seems unlikely.

If during the pause, the biceps has returned to resting level, then this interval can be regarded as akin to the time to initiate an extension movement from rest. In the absence of any effects of vision reducing IT of affected arm extension in Experiment 3.1, this seems unlikely to be the case. More probably the biceps has not fully relaxed and there may therefore be two barriers to extension commencing:-

1) The threshold necessary for triceps firing to effect movement is raised.

2) In attempting to achieve threshold, the triceps is hindered by inhibition from the mechanisms sustaining the tonic biceps activity.

In a similar fashion to the regulation of ongoing extension, vision could be acting to lower these barriers by modulating supraspinal impingement upon the spinal apparatus, informing the system that there is no movement when movement should be occuring.

This interpretation has to be considered in the light of performance of the other movements. Apart from unaffected arm flexion by EW, all the hemiparetic

subjects exhibit pauses before reversal with both arms. These results are somewhat surprising. It might have been expected that reversal of the affected arm from extension into flexion would proceed without pause due to stretch of the biceps on sudden release, evoking an enhanced reflex contraction, that would aid production of flexion. That it was found to be slower than normal, with pausing, might argue for a more symmetric dysfunction in reciprocal inhibition which makes the specific task of switching any synergies more difficult. However the finding that this reversal is not significantly different, in all patients, from reversal of the unaffected arm from flexion into extension, argues that if this is the case then the mechanism must be general, applying to both limbs.

In the absence of any published data supporting the idea that such a specific dysfunction occurs generally after stroke, this conclusion appears untenable. It is more probable that this finding can be linked with that of Experiment 3.1, that the unaffected arm takes longer to initiate movement and moves more slowly than normal. Both findings may be taken to reflect a general slowing down in CNS functioning following stroke.

To some extent reversal of the affected arm into extension must also be determined by any general lowering of speed of action. However it is argued that the

abnormal length of the pause interval and the finding that, alone amongst the other features of the movements, it can be visually reduced, points to a qualitative difference between this and other movements, as argued above.

## Experiment 3.4 - The influence of associated reactions on hemiparetic movement

### Introduction

This experiment was designed to study the effects of "associated reactions" (Walshe, 1923) on paretic arm performance. Walshe (1923) defined associated reactions as "released postural reactions in muscles deprived of voluntary control". They occur on exertion of activity in other parts of the body, e.g. in standing or on forceful contraction of the muscles of the unaffected arm and are held to result from "irradiation" of efference. This phenomenon, which induces changes of muscle tone, or indeed movement in the affected arm, has been widely reported by clinicians (Zulch and Muller, 1967). Indeed a recent survey by Mulley (1983) revealed that out of a sample of forty stroke patients, 80% had experienced associated reactions of one kind or another.

These reactions lie at the heart of the dispute between the Bobath (1978) and Brunnstrom (1970) regimes of physiotherapy. The Brunnstrom method uses them to elicit arm flexion when it is absent or weak, whilst followers of the Bobath method consider them detrimentally reinforcing to the development of a spastic pattern. Despite their widespread occurence and

potential importance to rehabilitation, no attempt has been made to study these reactions within the context of a voluntary movement of the affected arm. Rather, studies to date have investigated these reactions in the affected arm at rest. It has been implicitly assumed, but never empirically demonstrated, that associated reactions modify voluntary movement performance of the affected arm. Most studies (Zulch and Muller, 1967) have followed Walshe's (1923) line of investigation. A typical test in Walshe's study required the patient to lie supine on a bench with his arms hanging downward at each side. His unaffected hand held a rubber ball. This was squeezed and simultaneously the elbow was flexed. Walshe's data revealed that this procedure induced flexion in the paretic arm.

The experiment reported here is similar in procedure to the above example, except that in this study the affected arm performs a flexion or extension movement, instead of remaining at rest, whilst the subject exerts a tonic contraction of the unaffected wrist and elbow flexors. Although associated reactions are being investigated as a phenomenon in this experiment, the predicted reactions are being employed as a tool hypothesised to increase the flexor bias in the segmental structures underlying the control of the elbow joint. In doing this, it might be expected that there would be increased difficulty in performing extension, but

facilitation of flexion and thus a greater demand upon the processes governing visuo-motor control.

## Method

### Design

The task in this experiment was exactly the same as moving to the long distance target (L) of Experiment 3.1. The affected arm (AA) alone performed the task. A repeated measures design was employed with two factors:-

1) Direction (Dir) - Extension, Flexion.

2) Vision (Vis) - Full Vision (FV), No Vision (NV). This resulted in four conditions altogether with ten trials per condition.

### Subjects

The four hemiparetic subjects who took part in Experiment 3.1 also participated in this experiment.

### Apparatus

The same apparatus was used as in Experiment 3.1. In addition, a hand-grip was provided in an attempt to produce associated reactions.

### Procedure

For the same reasons as in Experiment 3.1, this experiment was not conducted within one session. The procedure was almost exactly the same as Experiment 3.1, but without UA and short distance target conditions.

In addition to moving the cradle with the affected

arm, subjects were required, immediately prior to moving, to squeeze the hand grip and simultaneously flex the elbow of the unaffected arm. They were instructed to do this just before the "Ready" signal preceding each trial and to maintain an isometric contraction until they judged attainment of the target distance by the affected arm.

The assessment of EMG resting levels of the triceps and biceps was sampled prior to running each condition, with the the unaffected arm in the state of isometric contraction described above.

Data Analysis

Analysis was exactly the same as for Experiment 3.1 and yielded the same five measures:-

1) Constant Error
2) Initiation Time
3) Movement Time
4) Number of submovements
5) Muscle Activity - a) Triceps, b) Biceps

Analysis of these measures was conducted individually for each subject using two-way, 2 x 2 (Direction x Vision) independent measures ANOVA's. Post-hoc comparison of individual means was by related t-tests.

## Results

### Constant Error

Means and standard deviations of constant error in each condition are tabulated in Table 3.4.1 for each subject, with the means plotted in Figure 3.4.1. The results of the ANOVA's performed on each subject's results are presented in Table 3.4.2.

The overall tendency found in all subjects across the visual conditions, is for extension movements to undershoot the target. Flexion movements are more accurate, across the visual conditions, and tend to overshoot in three of the subjects (JG, EW and MS) with the fourth (WI) erring slightly in the direction of an undershoot. Post-hoc analysis of the main effect of Dir found for all the subjects reveals these results to be significant.

Analysis of the main effect of Vis found for EW, WI and MS show it to be due to the degree of undershoot in the NV condition. However Figure 3.4.1. demonstrates that this result only holds for extension and this is supported by significant Dir x Vis interactions. JG's results run counter to this, with the significant two-way interaction resulting from an overshoot of flexion in the NV conditions.

Table 3.4.1

Mean (S.D.) of Constant Error

| CONDITIONS | | SUBJECTS MS | EW | WI | JG |
|---|---|---|---|---|---|
| | V | -0.21 | +0.63 | +0.41 | -0.06 |
| | | (2.46) | (0.75) | (1.09) | (1.39) |
| E | NV | -13.66 | -8.71 | -8.79 | -1.05 |
| ---------- | | (1.39) | (2.42) | (0.59) | (1.99) |
| F | V | +0.67 | -0.93 | -2.20 | +0.02 |
| | | (1.99) | (1.19) | (0.36) | (1.14) |
| | NV | +0.02 | +1.82 | +0.21 | +2.45 |
| | | (3.04) | (2.17) | (1.68) | (3.32) |

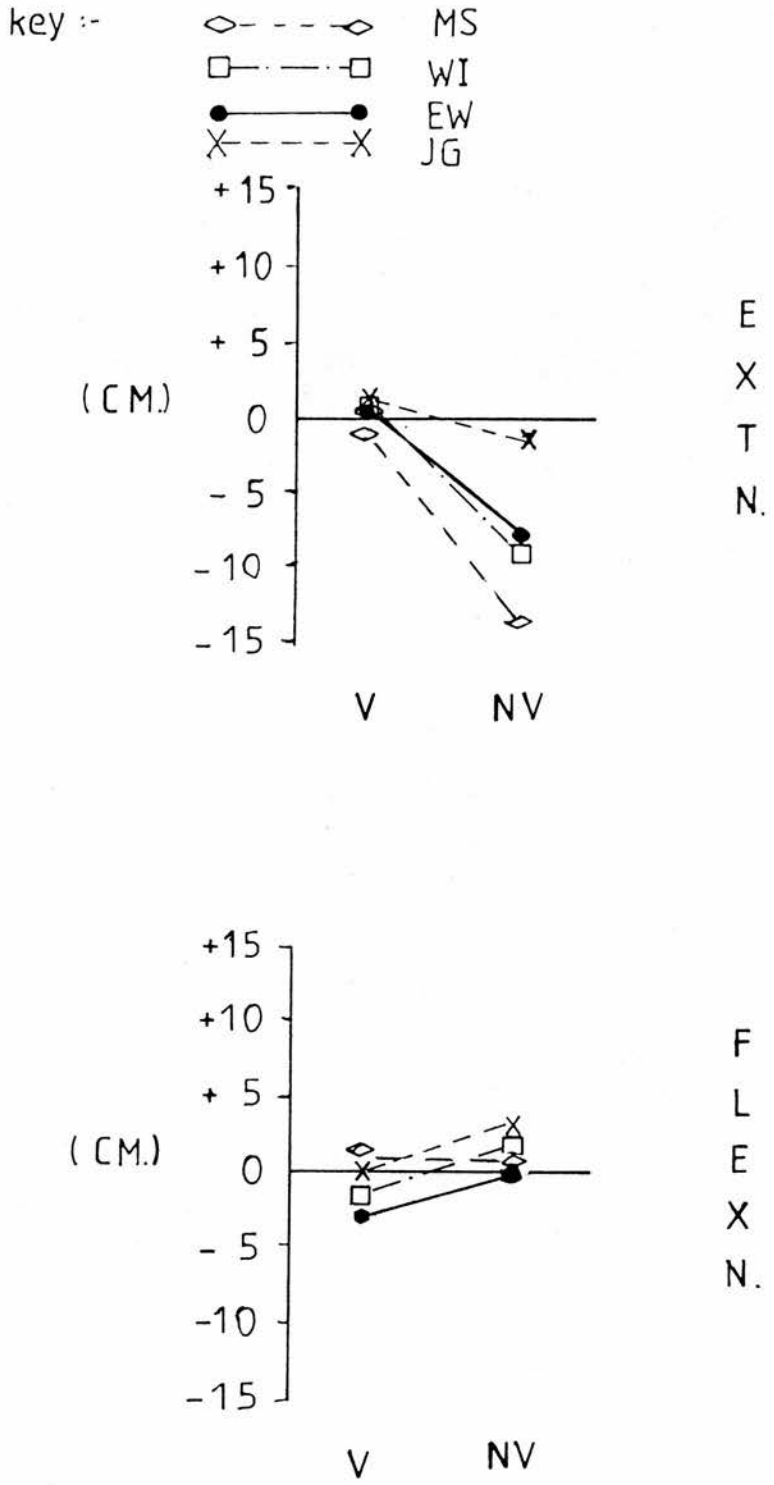



Figure 3.4.1.

Mean Constant Error.

Table 3.4.2

Anova Results for Constant Error

| SOURCE OF VARIATION | SUBJECTS | | | |
|---|---|---|---|---|
| DIR | 64.057 *** | 7.035 * | 100.055 *** | 90.365 *** |
| VIS | 34.574 *** | 1.138 ns | 93.832 *** | 102.033 *** |
| DIR X VIS | 116.369 *** | 6.420 * | 77.328 *** | 298.308 *** |

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

ns non-significant

Initiation Time

Means and standard deviatiions of IT are presented in Table 3.4.3 and the ANOVA results are listed in Table 3.4.4. Figure 3.4.2, which illustrates each subject's mean results, shows a general trend for IT to be shorter under NV conditions. However this is only significant for one subject (MS), as indicated by the main effect of Vis.

The only other difference on IT across the conditions was for Dir, with main effects of this factor (JG and WI) indicating flexion movements commenced sooner after the stimulus to move.

Table 3.4.3

Mean (S.D.) of Initiation Time (secs)

| CONDITIONS | | SUBJECTS MS | EW | WI | JG |
|---|---|---|---|---|---|
| | V | 2.940 | 1.031 | 1.269 | 1.488 |
| | | (1.083) | (0.515) | (0.693) | (0.750) |
| E | NV | 1.903 | 0.721 | 1.038 | 1.193 |
| | | (0.916) | (0.207) | (0.474) | (0.645) |
| F | V | 2.575 | 0.912 | 0.695 | 0.740 |
| | | (0.678) | (0.597) | (0.511) | (0.468) |
| | NV | 1.319 | 0.738 | 0.297 | 0.893 |
| | | (0.354) | (0.197) | (0.127) | (0.105) |

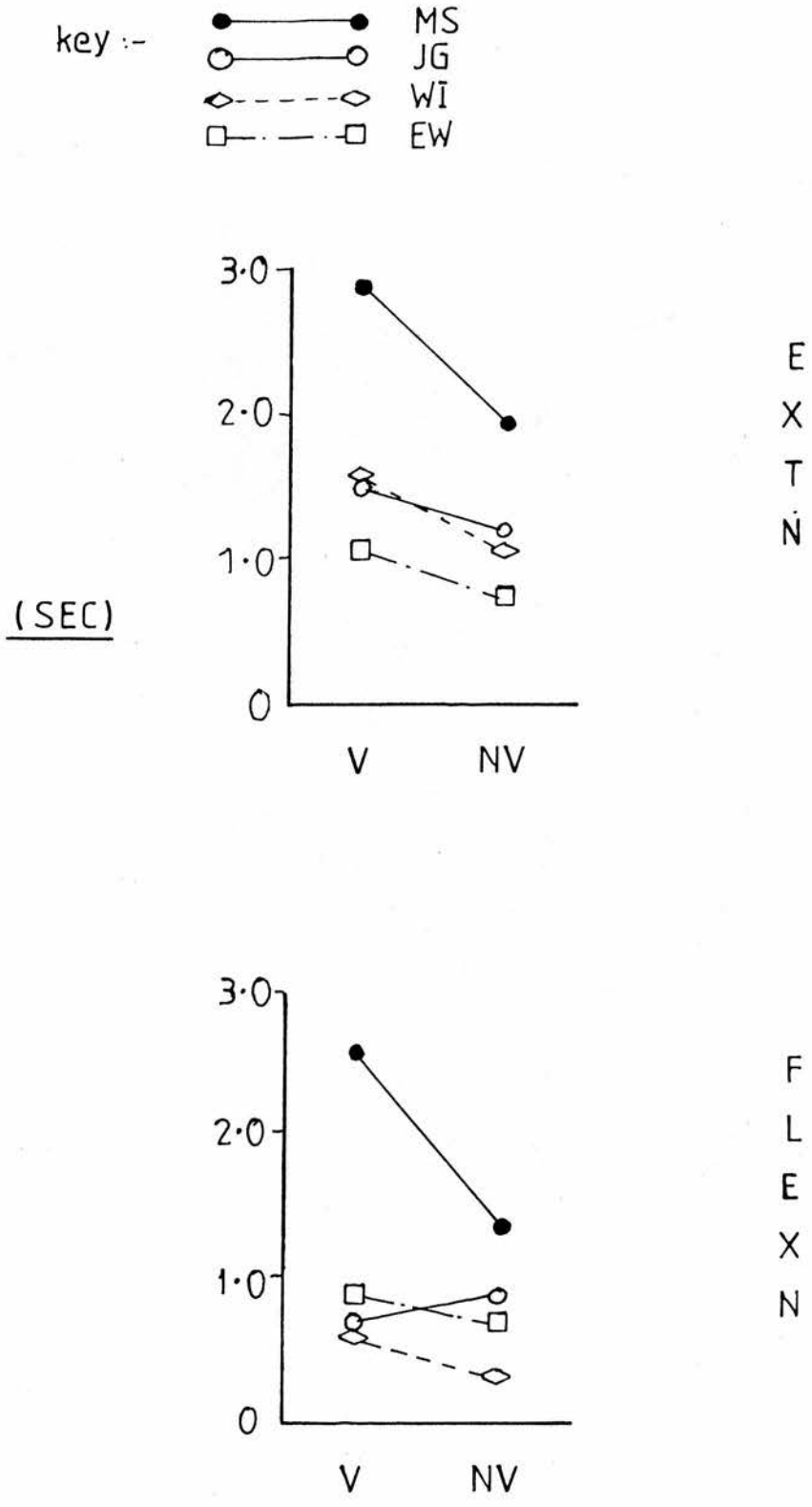



Figure 3.4.2. Mean Initiation Time.

Table 3.4.4

Anova Results for Initiation Time

| | SUBJECTS | | | |
|---|---|---|---|---|
| SOURCE OF VARIATION | EW | JG | MS | WI |
| DIR | 0.148 | 9.073 | 3.468 | 17.580 |
| | ns | ** | ns | *** |
| VIS | 3.328 | 0.167 | 20.247 | 4.022 |
| | ns | ns | *** | ns |
| DIR X VIS | 0.263 | 1.658 | 0.185 | 0.284 |
| | ns | ns | ns | ns |

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

Movement Time

Means and standard deviations of MT are presented in Table 3.4.5. However, as in Experiment 3.1, because of differences in the distance moved across conditions, mean velocity was calculated and taken to represent the subject's ability to quickly move to the target.

Velocity - Means and standard deviations of velocity are documented in Table 3.4.6 with the means illustrated in Figure 3.4.3. The results of the ANOVA's performed on each subject's data are presented in Table 3.4.7.

Post-hoc analysis of the main effect of Dir found for each subject's data, show it to result from flexion movements being faster than extension movements. A main effect of Vis for JG and WI are due to NV conditions producing a faster movement. As Figure 3.4.3 shows, this result only holds true for flexion movements. Significant Dir x Vis interactions support this conclusion.

The significant main effect of Dir found for MS is qualified by a two-way interaction which shows it due to faster extension performance when sight of the arm is permitted.

Table 3.4.5

Mean (S.D.) of Movement Time

| CONDITIONS | | SUBJECTS MS | EW | WI | JG |
|---|---|---|---|---|---|
| E | V | 2.877 | 7.244 | 5.205 | 2.659 |
| | | (1.251) | (2.153) | (1.090) | (0.751) |
| | NV | 1.747 | 4.519 | 2.938 | 2.763 |
| | | (0.503) | (1.317) | (0.661) | (0.936) |
| F | V | 1.886 | 3.145 | 5.111 | 3.711 |
| | | (0.659) | (1.424) | (0.858) | (0.357) |
| | NV | 1.533 | 3.261 | 3.021 | 2.012 |
| | | (0.420) | (0.730) | (0.977) | (0.671) |

Table 3.4.6

Mean (S.D.) of Velocity (cm/sec)

| CONDITIONS | | SUBJECTS MS | EW | WI | JG |
|---|---|---|---|---|---|
| | V | 7.93 | 3.10 | 4.11 | 8.07 |
| | | (2.86) | (1.03) | (1.03) | (2.47) |
| E | NV | 4.21 | 2.62 | 3.98 | 7.44 |
| ---------- | | (2.51) | (0.65) | (0.89) | (2.18) |
| F | V | 12.17 | 6.90 | 3.59 | 5.43 |
| | | (4.35) | (2.14) | (0.73) | (0.70) |
| | NV | 13.84 | 6.99 | 7.35 | 12.16 |
| | | (3.60) | (1.60) | (2.43) | (4.32) |

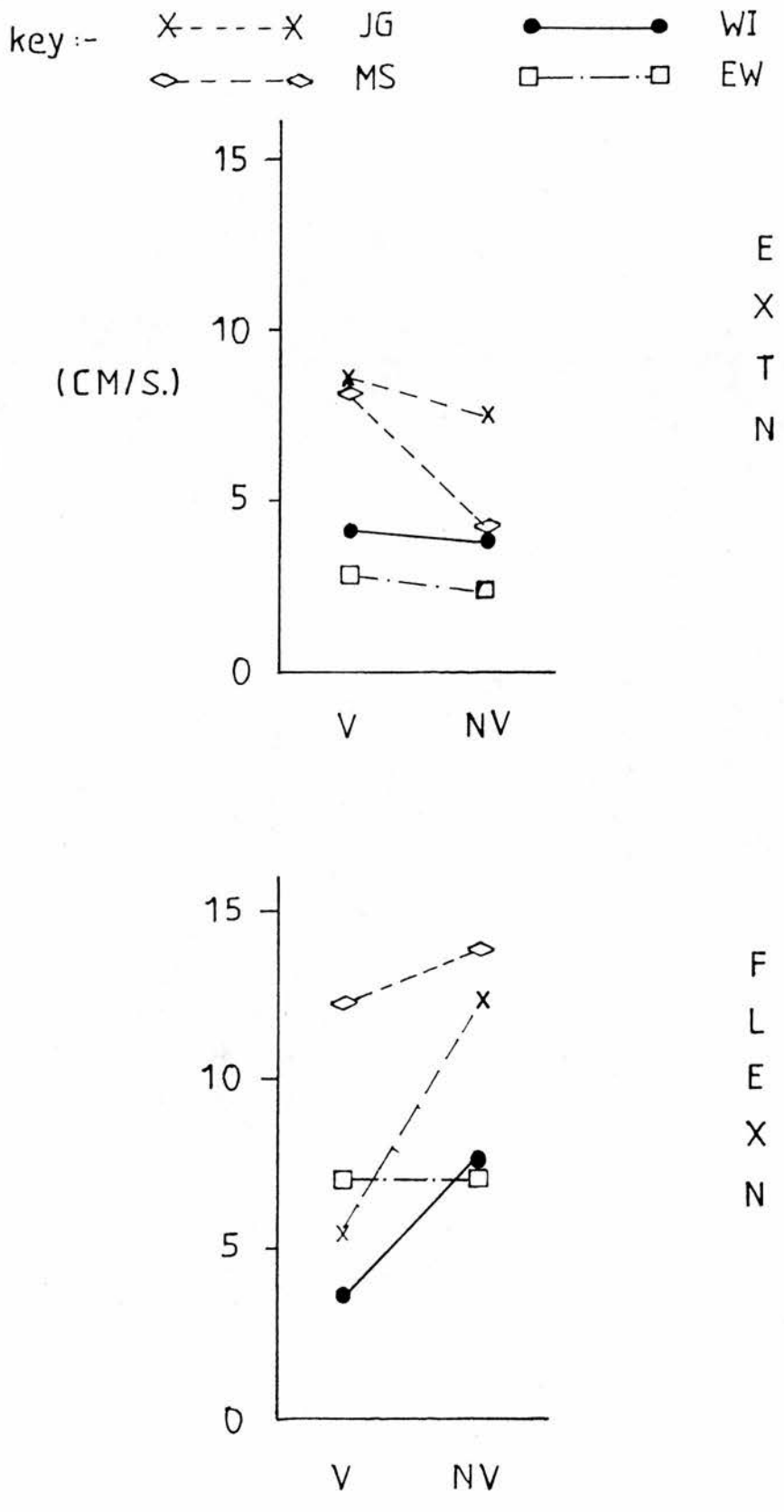



Figure 3.4.3. Mean Velocity.

Table 3.4.7

Anova Results for Velocity

| SOURCE OF VARIATION | SUBJECTS EW | JG | MS | WI |
|---|---|---|---|---|
| DIR | 77.251 *** | 1.442 ns | 41.523 *** | 9.815 ** |
| VIS | 0.176 ns | 12.399 ** | 0.908 ns | 15.923 *** |
| DIR X VIS | 0.376 ns | 18.051 *** | 6.274 * | 18.286 *** |

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

ns non-significant

Number of Submovements

Means and standard deviations of the number of submovements present in each subject's task trajectories are displayed in Table 3.4.8. However as in Experiment 3.1, to account for differences across conditions in the distance encompassed the data were converted to mean distance traversed within one submovement.

Mean Submovement Distance - Means and standard deviations of this measure are presented in Table 3.4.9 with the means plotted in Figure 3.4.4. The results of the ANOVA's are given in Table 3.4.10.

A main effect of direction of movement was found for EW, WI and MS with post-hoc analysis revealing submovements composing flexion to cover a greater distance than those composing extension. This can be clearly seen in Figure 3.4.4 which illustrates opposite trends in the two movements when sight of performance is withdrawn. In extension movements, the mean submovement distance decreases, whilst in flexion it increases. This is reflected in the significant two-way interactions found for all subjects. Analyses of these interactions reveal, in each case, that significance is solely due to the pattern of results for flexion movements.

Table 3.4.8

Mean (S.D.) of Number of Submovements

| | | SUBJECTS | | | |
|---|---|---|---|---|---|
| CONDITIONS | | MS | EW | WI | JG |
| | V | 5.10 | 7.70 | 7.50 | 5.20 |
| | | (1.60) | (2.26) | (1.27) | (0.92) |
| E | NV | 2.60 | 4.90 | 4.30 | 6.00 |
| | | (0.97) | (0.88) | (0.82) | (1.56) |
| F | V | 3.00 | 4.60 | 4.80 | 7.70 |
| | | (1.05) | (0.84) | (1.55) | (1.06) |
| | NV | 2.10 | 3.70 | 2.80 | 4.90 |
| | | (0.57) | (0.48) | (0.92) | (1.37) |

Table 3.4.9

Mean (S.D.) of Submovement Distance (cm)

| | | SUBJECTS | | | |
|---|---|---|---|---|---|
| CONDITIONS | | MS | EW | WI | JG |
| | V | 4.30 | 2.97 | 2.79 | 3.93 |
| | | (1.74) | (1.16) | (0.55) | (0.82) |
| E | NV | 2.87 | 2.33 | 2.71 | 3.27 |
| --- | | (1.49) | (0.43) | (0.49) | (0.59) |
| F | V | 7.69 | 4.24 | 4.23 | 2.63 |
| | | (2.85) | (0.68) | (1.90) | (0.39) |
| | NV | 10.18 | 5.96 | 8.45 | 4.97 |
| | | (3.04) | (0.68) | (4.67) | (1.83) |

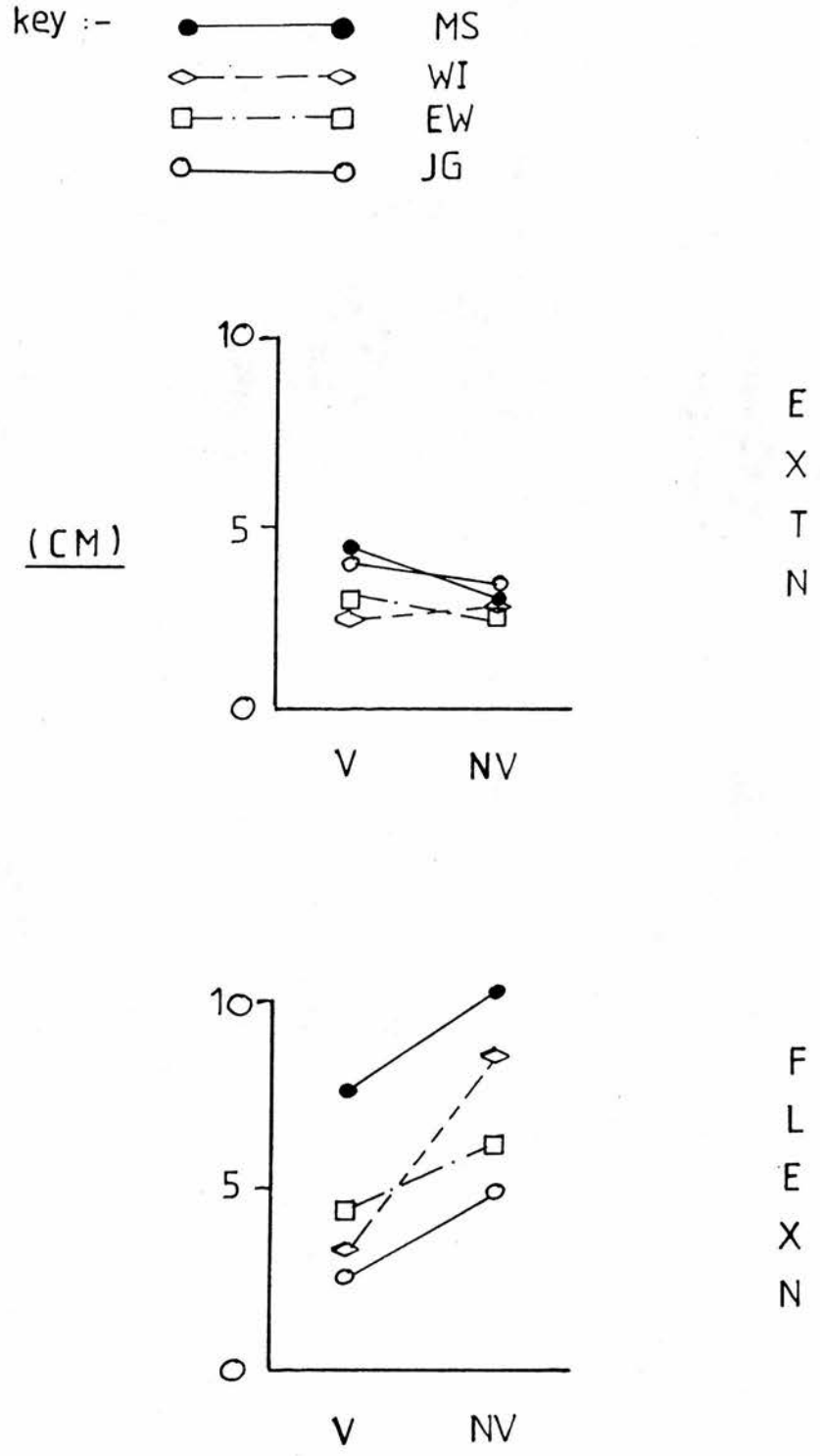



Figure 3.4.4. Mean Sub-Movement Distance.

Table 3.4.10

Anova Results for Mean Submovement Distance

| | SUBJECTS | | | |
|---|---|---|---|---|
| SOURCE OF VARIATION | EW | JG | MS | WI |
| DIR | 98.267 | 0.353 | 50.608 | 19.860 |
| | *** | ns | *** | *** |
| VIS | 4.774 | 6.222 | 0.497 | 6.603 |
| | * | * | ns | * |
| DIR X VIS | 22.795 | 19.841 | 6.792 | 7.123 |
| | *** | *** | * | * |

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

ns non-significant

Muscle Activity

Biceps - Means and standard deviations of the percentage of the movement time for which the biceps was active are given in Table 3.4.11 with the means plotted in Figure 3.4.5. The ANOVA results are presented in Table 3.4.12.

All subjects exhibit a main effect of Dir due to biceps being more active during flexion. This is clearly shown in Figure 3.4.5.

Biceps - Means and standard deviations of the percentage of the movement time for which the biceps was active are given in Table 3.4.11 with the means plotted in Figure 3.4.5. The ANOVA results are presented in Table 3.4.12.

All subjects exhibit a main effect of Dir due to biceps being more active during flexion. This is clearly shown in Figure 3.4.5. There is a tendency in extension movements for activity to be higher under NV and the main effect of vision with significant two-way interactions, demonstrate this trend to be significant for EW and MS.

Triceps - Means and standard deviations of the measure of activity for this muscle are presented in Table 3.4.13 with the means illustrated in Figure 3.4.6 and the ANOVA results tabulated in Table 3.4.14.

A main effect of Dir is exhibited by all subjects with as Figure 3.4.6 shows, higher activity during extension. The only other significant result was for JG

with a main effect of Vis and a significant two-way interaction, indicating triceps activity to be higher in flexion under NV.

Table 3.4.11

Mean (S.D.) of Biceps Activity

| | | SUBJECTS | | | |
|---|---|---|---|---|---|
| CONDITIONS | | MS | EW | WI | JG |
| | V | 70.60 | 60.31 | 70.52 | 74.62 |
| | | (17.90) | (16.09) | (11.15) | (15.11) |
| E | NV | 92.17 | 93.33 | 80.46 | 80.59 |
| | | (9.24) | (8.78) | (15.36) | (13.77) |
| F | V | 97.03 | 99.11 | 98.84 | 98.21 |
| | | (9.39) | (2.81) | (3.66) | (2.74) |
| | NV | 100.00 | 100.00 | 99.66 | 100.00 |
| | | (0.00) | (0.00) | (1.08) | (0.00) |

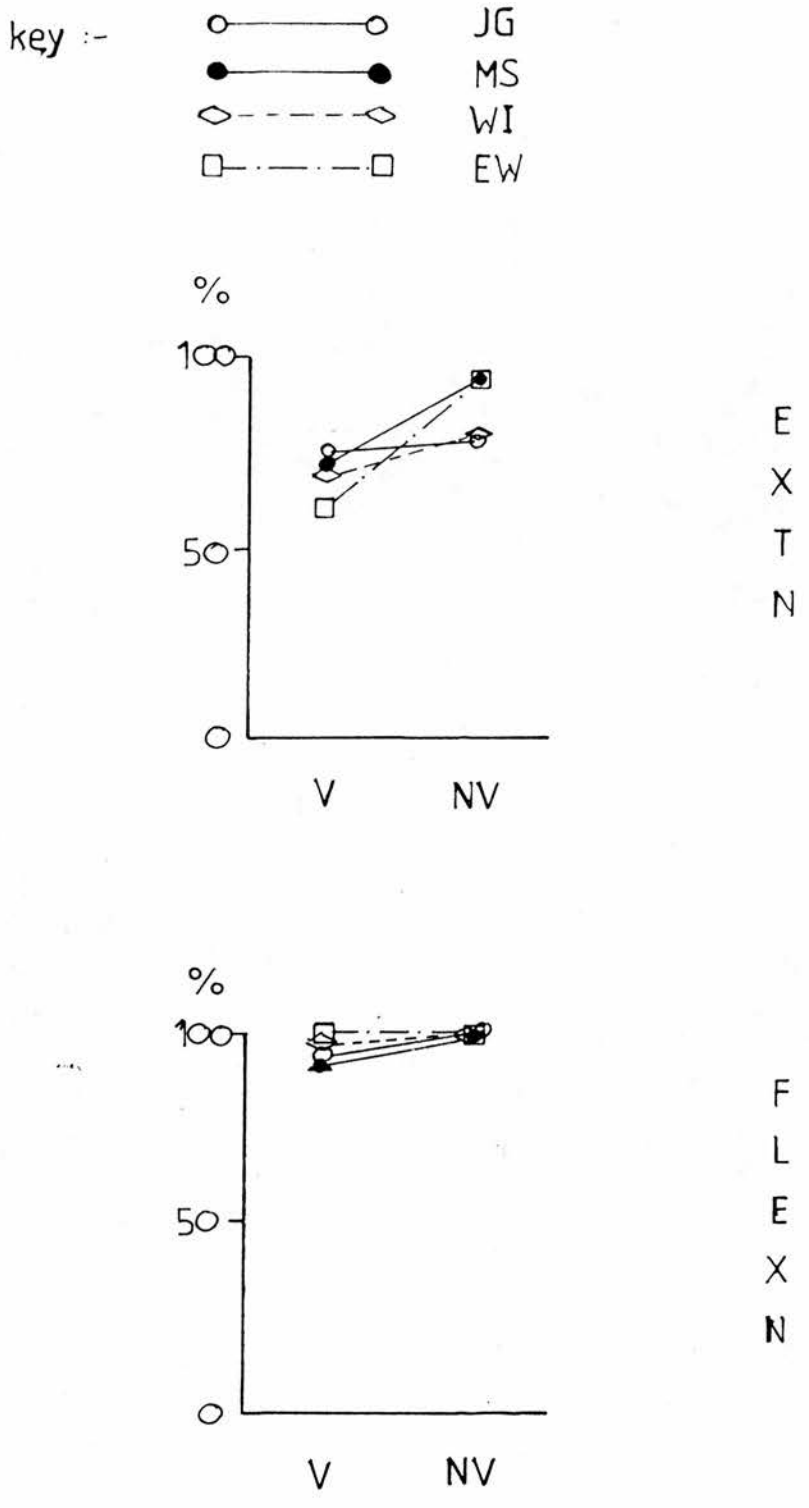



Figure 3.4.5.

Mean Biceps Activity.

Table 3.4.12

Anova Results for Biceps Activity

| SOURCE OF VARIATION | SUBJECTS | | | |
|---|---|---|---|---|
| DIR | 60.106 *** | 43.475 *** | 23.765 *** | 60.251 *** |
| VIS | 33.429 *** | 0.411 ns | 12.193 ** | 2.219 ns |
| DIR X VIS | 30.012 *** | 1.416 ns | 7.005 * | 3.089 ns |

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

ns non-significant

Table 3.4.13

Mean (S.D.) of Triceps Activity

| CONDITIONS | | SUBJECTS EW | JG | MS | WI |
|---|---|---|---|---|---|
| | V | 100.00 | 99.46 | 100.00 | 100.00 |
| | | (0.00) | (1.71) | (0.00) | (0.00) |
| E | NV | 100.00 | 96.22 | 100.00 | 100.00 |
| --- | | (0.00) | (8.22) | (0.00) | (0.00) |
| F | V | 11.68 | 0.00 | 0.00 | 0.00 |
| | | (18.52) | (0.00) | (0.00) | (0.00) |
| | NV | 0.00 | 0.00 | 0.78 | 12.36 |
| | | (0.00) | (0.00) | (2.47) | (16.06) |

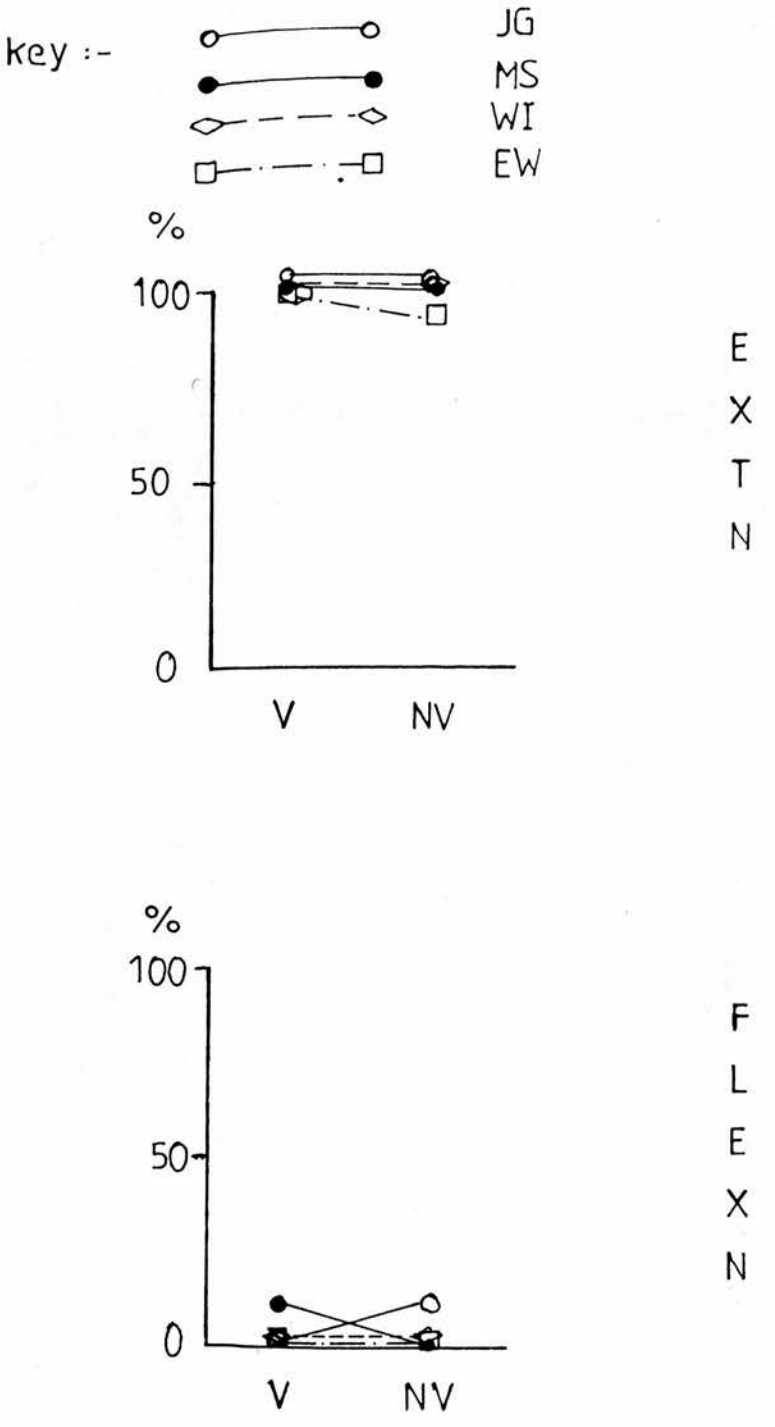



Figure 3.4.6.

Mean Triceps Activity.

Table 3.4.14

Anova Results for Triceps Activity

| SOURCE OF VARIATION | SUBJECTS EW | JG | MS | WI |
|---|---|---|---|---|
| DIR | 5429.074 *** | 1364.726 *** | 1033.950 *** | 65234.387 *** |
| VIS | 1.486 ns | 5.921 * | 3.977 ns | 1.000 ns |
| DIR X VIS | 1.486 ns | 5.921 * | 3.977 ns | 1.000 ns |

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

ns non-significant

## Discussion

Although this experiment is presented fourth in this Chapter, it was in fact run immediately after Experiment 3.1, and had the same subjects. The idea at the time was that a direct comparison could then be made between the conditions of this experiment, with the same conditions, minus accompanying unaffected arm activity, in Experiment 3.1. However the running of the two experiments was typically spread over a period of around two weeks for each subject. During that period it is possible that changes took place in the patients' motor function. The only check made on this was to compare the assessments made by the physiotherapist treating each patient, before and after this period. In all cases they reported negligible alteration in arm function.

Nevertheless more subtle changes in motor performance could have taken place which may have affected the outcome of this experiment. This therefore reduces the utility of any precise statistical comparison. For this reason, less exacting comparisons and contrasts are made between the two experiments, on the assumption that any consistent similarities or differences found are truly due to experimental manipulation, since the individually different random order of presentation of conditions in both experiments may have cancelled the effect of any temporal changes.

In general the pattern of results follows that of Experiment 3.1. Flexion movements are faster and cover a greater distance per sub-movement. Additionally, in flexion, the antagonist triceps is minimally active, whilst in extension the biceps is highly active. The following discussion focuses on the two questions central to this experiment: does a flexor contraction in the unaffected arm facilitate affected arm flexion and conversely does it inhibit extension?

As regards the first point, the mean overall speed of flexion is not too different from Experiment 3.1. There is however a more significant separation between the the two visual conditions in this experiment. Three of the subjects show a reduction in velocity when visual monitoring is permitted. A similarly enhanced direction of separation is also apparent in the mean sub-movement data with fewer sub-movements when vision is withdrawn. However, overall, the mean sub-movement distance has not increased. Therefore taking these two measures as the primary indices of ease of movement, it can be concluded that any associated reactions present have not facilitated flexion.

This then begs the question of whether associated reactions were indeed present in this experiment. Turning to the extension and initiation time data provides an answer.

If they are present then a rise in biceps activity might be expected during extension. This is clearly the case. Notably, comparison of Figure 3.1.9 with Figure 3.4.5, demonstrates a less significant difference in biceps activity across the two visual conditions. Following from the arguments of Experiment 3.1, does this reflect in poorer extension performance? Certainly overall mean velocity is reduced in this experiment, but this is principally due to a lowering of speed in the FV condition as demonstrated by only one subject showing a significant drop from FV to NV. Similarly on mean submovement distance, in contrast to Experiment 3.1, no significant difference was found across the visual conditions.

Therefore the primary effect on affected arm extension is that the improvement gained in smoothness and speed of movement through visual monitoring is reduced when the unaffected arm is in a state of forceful isometric flexion. The EMG data suggest that even with the aid of visual monitoring the increased flexor bias imposed on the control of the elbow cannot be combatted by voluntary control. However despite the reduction in movement quality, accuracy of performance under FV is maintained. This demonstrates that the other two proposed functions of vision still operate with regard to perception of limb position and appreciation of effort.

One of the major differences between the two experiments is on initiation time. Overall in this experiment it was found to be higher. At the time of running it was noticeable that subjects appeared to have greater difficulty in commencing movement. This finding may be better appreciated by considering the procedure adopted here in the context of previous studies. All prior work on this form of associated reaction has studied the elicitation of movement in the affected arm when it was more plegic than paretic (Walshe, 1923; Brunnstrom, 1970). It is arguable that the patients studied here had a greater degree of voluntary control. This highlights the second procedural difference. The patients were attempting to make a movement to a specified target which they already knew they could perform. Therefore this procedure encouraged a controlled volition. If the isometric contraction in the unaffected arm biased the spinal apparatus in the direction of flexion prior to the imperative signal to move then the increase in IT for extension probably reflects the additional task of trying to control for or counter this bias.

It might be expected that compared to Experiment 3.1, IT for flexion would fall because of the additional bias for flexion. This was not the case and could indicate that similar to preparation for extension, initiation of flexion attempts to control for the bias,

even although it would favour a speedy production of movement. Some of the other results for flexion hint at this control operating during execution. All subjects have a significantly lower mean sub-movement distance under FV. In addition two of them move with a significantly lower mean velocity. It may be that with an increased bias towards flexion, vision is used to try prevent ballistic uncontrolled movement.

Despite the time difference between the two experiments some confidence can be expressed in the conclusions derived from their comparison. This is because the results demonstrate differential significant interactions, in addition to differences in absolute mean values. The fuller relevance of the results to hemiparetic motor control is discussed in the next section.

## General Discussion

The aim of this Chapter was, through kinematic analysis, to elucidate differences in motor performance between the affected and unaffected arms, and to compare this with movement of the normal arm. In doing so, it was supposed that a clearer understanding of the principal result of Chapter II would ensue, ie.that when sight of performance is withdrawn extension of the affected arm undershoots its goal. As the results of Experiment 3.1 demonstrate, this proved to be the case. The main result of that experiment was the highlighting of differences between affected arm extension and flexion as compared with unaffected and normal performance. Extension is more discontinuous and slower, with an abnormally high amount of antagonist activity. Moreover, extension, but not flexion deteriorates when sight of performance is withdrawn.

Following these results, three possible roles of visual information in the control of affected arm extension were postulated:-

1) The correction of perception of degree of elbow extension, misinformed by hyperactivity in the antagonist biceps afferents.

2) A related correction through a realistic appreciation of the amount of effort required to achieve a target joint angle.

3) An increased ability to switch off inappropriate antagonist biceps co-contraction.

All these hypotheses have received some, but by no means conclusive, support from the experiments contained in this Chapter. In discussing the implications of these hypotheses in hemiplegic motor control the salient results of the Chapter are discussed, somewhat speculatively, with what knowledge exists concerning dysfunction at the spinal level. One model of spinal functioning, derived from Tanaka (1974) and Yanagisawa et al (1976), is presented in Figure 3.2.

The alpha and gamma motoneurons with the corresponding Ia interneuron are considered as a functional unit (Lundberg, 1971; Hultborn, 1972), under supraspinal modulation, that governs contraction of the agonist and relaxation of the antagonist. Evidence from several studies (pp. 13-22) suggests that in hemiparesis, concurrent with an increase in monosynaptic relex excitability, which reflects hyperexcitability in the alpha motoneuron, there is increased excitation of the corresponding gamma system and Ia interneurons.

This increase in excitability is hypothesised to be due to a release of these structures from inhibitory supraspinal control. However although Tanaka (1974) demonstrated hyperexcitability in both the hypertonic and hypotonic members of an agonist/antagonist linkage,

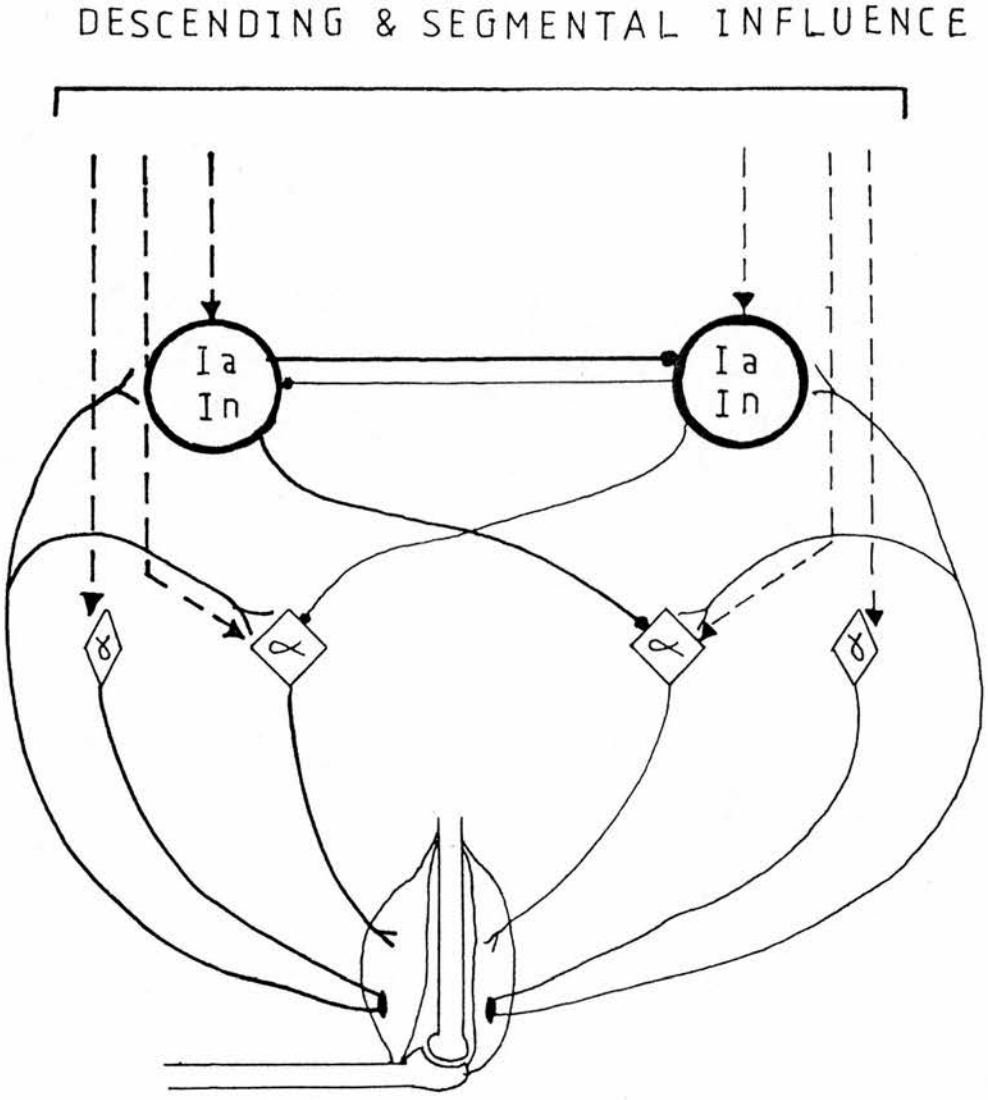



Figure 3.2 Spinal connections.
(after Yanagisawa et al(1976)).

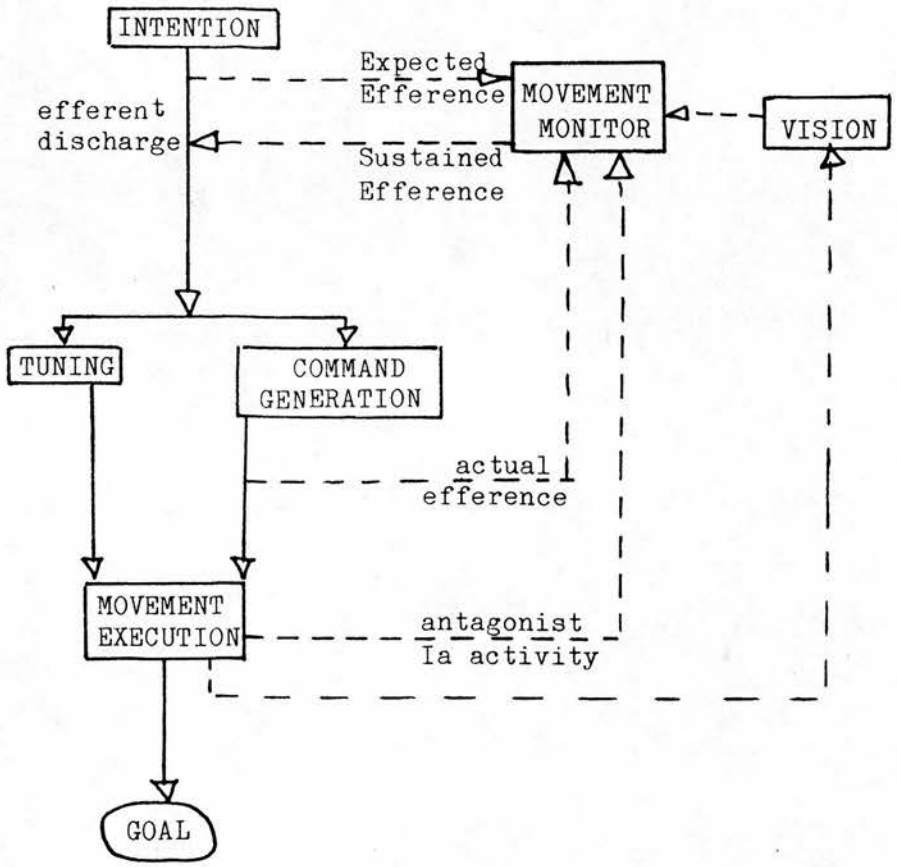



Figure 3.3 - Tentative model of hypothetical processes underlying affected arm extension.

Yanagisawa et al (1976) postulate that release from inhibition of the Ia interneurons is predominant in those composing the functional unit of the hypertonic flexor muscle. This is reflected in the Figure by the difference in thickness between the descending interrupted lines.

It is uncertain how this imbalance in supraspinal projection comes about. Studies of locomotion in the cat have revealed that different descending pathways exert different effects on hindlimb interneurons. For example, Orlovsky (1972) has shown that the reticulo-spinal tract exerts an excitatory influence on flexors and inhibits extensors, whilst the vestibulo-spinal tract excites only extensors. Therefore at the segmental level there is normally a fine interplay of excitatory and inhibitory projections determining eventual muscle activity. By some as yet to be elucidated process this dynamic and fluid interaction is lost following stroke. The effect of release therefore causes the thicker lined spinal connections to be predominantly active. Following through the model, some of the mechanisms postulated as underlying affected arm extension can be traced.

During the initiation period of extension the increased excitation of the flexor structures causes difficulties in both firing the extensor alpha motoneurons and in inhibiting the flexor alpha's. During

execution the extensor structures have to compete against inhibition from flexor Ia interneurons fuelled by enhanced Ia afferent activity which also fires flexor alpha motoneurones. This would explain the high amount of biceps activity found during extension and also the slow halting nature of the movement.

These events at the spinal level can be transposed to a more global model of extension performance which is presented in Figure 3.3. To introduce the model, performance of a normal movement is considered first. Through prior instruction the subject intends to extend the arm from a seen starting position to a visually specified goal. On receipt of the imperative stimulus, that intention or plan for action is translated into two processes (Kots, 1977). Firstly the spinal structures are tuned in order to bias the limb for the intended movement. An important component of this process is a lowering of excitation in the antagonist motoneuron pool and an appropriate adjustment of the relative gains of the Ia inhibitory pathways. Simultaneous with tuning, commands are generated to activate the particular motoneuron pools necessary to produce contraction in the appropriate agonist. On activation, execution of movement commences. As the results of the normal subject (AL) in Experiment 3.1 demonstrated, the intact human system is capable of successfully executing the movement largely on the basis of pre-movement neural control.

However as Bernstein (1967) indicated, pre-planning cannot deal with unexpected perturbation interfering with performance. Therefore some sort of back-up monitoring process of the course of movement is necessary to achievement of the precise goals of the task. Whilst it is acknowledged that the majority of normal movement can proceed on a pre-planned basis, even the most vociferous proponents of pre-planning admit that the potential exists for correction via ongoing monitoring. This is necessary to explain the existence of fast, fine correction in the latter stages of movement. In Experiment 3.1, AL demonstrated that in the normal performance of a discrete movement somatically based information, although slightly more uncertain, is equivalent to visually registered information in dictating how the fine correction should be made. It has been empirically demonstrated that both efferent (Angel, 1976) and afferent (Capaday and Cooke, 1981), sources of information pertaining to the course and likely success of movement are monitored during execution. In the current model a feedback of efferent activation of the agonist motoneurons and resultant Ia activity from the antagonist are postulated as important contributions to the flow of information to the monitoring process. When the task can be seen vision can also serve as an informant channel.

In normal movement therefore, prior tuning and

appropriate generation of motor commands largely determine execution. However as a contingency for perturbation or error, a process exists which compares intended effort with actual efference, somatically based proprioception, and visual registration of the course of the movement. It is suggested here that in the instance of affected arm extension this process is not merely a contingency, but plays a dominant role in motor control.

Consider first the case of extension when visual monitoring is not permitted. In the translation from intention to tuning and command generation, the expected effort registered by the monitor could underestimate the difficulties the movement will encounter. Experiments 3.1 and 3.4 demonstrated that the processes of tuning and command generation seem inefficient in programming movement. During execution, the inability to switch off antagonist activity combats the effectiveness of the agonist drive, and at the same time Ia activity in the antagonist informs the monitoring process that the arm is further extended than it really is. Simultaneous with this, matching of efference delivered to the agonist with expected effort, informs the system that the movement is proceeding along intended lines. In the microstructure of the movement these events conspire to produce discontinuity in the trajectory. At the macro level, movement is slowed down and the goal undershot. In both cases commands from the monitoring process to sustain

efferent drive to the agonist are reduced.

When the movement can be visually monitored, this affords an additional flow of information to the monitor. It permits the system to realise that despite somatic information to the contrary, performance is not as was intended. Slowing of movement is better registered and efferent activity sustained, both resulting in less discontinuity. It is also directly obvious if and when the target is reached, thus efference is sustained at the macroscopic level to achieve the goal. From the results of Experiment 3.4 it is possible that the mechanisms sustaining efference at the two levels may be different. Identification of these processes, whether disinhibitory or directly facilitatory, awaits more sophisticated electrophysiological examination. In summary, the system cannot depend on pre-tuning when extending the arm. Therefore in a sense the patient has to continually tune his movement as it is progressing and vision provides an essential source of information for him to do this.

Although the above account is somewhat speculative it does concur with the results obtained in this Chapter for extension of the affected arm. However there appears to be a mismatch between the neurophysiological model presented in Figure 3.1 and the results flexion of the affected arm. In Experiments 3.1 and 3.4, it was expected but not observed that, in the absence of visual

monitoring, flexion would be ballistic and would overshoot the target, due to hyperexcitability in the flexor structures. Failure to verify this forces the conclusion that, in this sample of patients at least, hyperexcitabilty was controlled during flexion. The results of Experiment 3.4 indicate that even when the flexor bias is increased it can be controlled. The finding that in these circumstances vision modulates flexion by making it slower and more discontinuous, but has a reduced power to affect control of extension, emphasises the disparity between the control mechanisms underlying the two movements.

In total, the results seem to indicate that the principal dysfunction following stroke lies in modulation of interneuronal processes by supraspinal influences. Quite precisely how control is exercised is at present unknown. The present results point to a complex interaction involving many levels of the CNS which can only be elucidated by studying the neurophysiology of hemiparesis with a combination of sophisticated physiological and behavioural techniques.

Before closing this Chapter a word needs to be said about the quality of unaffected, compared to normal, movement. It was consistently found that the so-called unaffected arm did not match the quality of performance exhibited by the normal control. Generally, von

Monakow's concept of diaschisis is invoked to account for this. In reviewing studies of recovery from focal lesions resulting from combat injuries, Teuber (1975) reports that there is typically an "overshoot" effect into general intellectual functioning. That is to say that whilst it might be expected that a lesion would affect specific functions, according to its site, it has been found that it also interferes with more general functions such as scores on IQ and reaction time tests (Teuber, 1975). The basic idea underlying this concept is that since the CNS is a functionally integrated system, damage to any particular part is bound to produce a lesser, widespread dysfunction, in addition to the resultant acute and specific disorder. This may underly the hypothesis of a general slowing down of function, evoked in this Chapter to explain some of the results.

The fact that this problem has not been clinically reported is probably a reflection of the manner in which the patients are normally assessed. Typically, clinical tests to diagnose stroke and assess recovery, focus on the abnormalities that characterise hemiplegia and hemiparesis such as hypotonus or hyperactive reflexes and they do not search for less obvious, general dysfunction.

# CHAPTER IV

## Interaction Between The Two Arms

## Introduction

Recently, there has been renewed interest in the use of bilateral co-ordination as a paradigm for the study of the processes underlying the organisation for normal motor control (Kelso et al, 1979; Klapp, 1979; Martenuik and McKenzie, 1980). Kelso et al (1979) employed a target aiming task in which movements were carried out by the right and left arms either individually or in unison to near/wide targets or far/narrow targets. With single arm movements, Kelso et al found that movement times were consistent with Fitt's Law (Fitts, 1954): movement time to near / wide targets was less than to far / narrow targets. The result for double arm movements to the same width of target, over identical distances, whilst showing a modest increase in initiation time and movement time over single arm movement, also complied with the Law when the movement times to the two target types were compared. However, in double conditions where the arms were moving to different targets types, the duration of the easier movement increased to that of the more difficult movement. Moreover, the entire temporal pattern of the course of the easy movement matched its difficult counterpart. Thus, overall the timing for both arms was dictated by the greater level of difficulty.

In a similar study, Martenuik and McKenzie (1980)

examined the effect of varying distance to target and mass at limb extremity in double arm movements. They replicated Kelso et al's finding that double movements to easy targets were executed more rapidly than to difficult ones. However, in contrast to Kelso et al, when the task demands were different for each arm, instead of the arm performing the easy task being compromised by and matching the performance of the difficult task, an interference or consensus effect occurred. Comparison of activities in differential double arm conditions to individual arm performances in double conditions, where the tasks were identical, revealed that the movement time of the easy task increased and the movement time of the difficult task decreased. Therefore there was a detrimental effect on the arm performing the easy task and a facilitation effect on the arm carrying out the difficult task.

The experiments to be reported here are similar to the above. However, with hemiparetic subjects it would be unrealistic to design a study where levels of ease or difficulty of task could be equated across the two arms. Of interest here therefore, is the performance of both arms when they are moving over identical distances to individual targets of equal size, ie in the execution of equivalent tasks.

The first question raised by this situation is what

might the hemiparetic subjects do spontaneously in this situation. The arms might move completely independently, with each action being planned and executed individually. Given the results of Experiment 3.4, this would be unlikely to be indicative of loss of communication between the processes governing the movement of each arm. Rather, it would imply that the patient had adopted a strategy designed not to compromise healthy limb performance, through choosing not to attempt to temporally couple the two actions together. Since pilot work indicated that this was unlikely to be the case, consideration of what might ensue when the arms are moving at or around the same time poses a more interesting question.

There are two possible hypotheses regarding the outcome.

1) The compromise hypothesis: - Following the results of Kelso et al (1979) the movement of the normal arm (easy task) would compromise to the action of the affected limb (difficult task) i.e. it might mimic its hemiparetic counterpart.

2) The consensus hypothesis: - From Martenuik and McKenzie's study there could be a consensus of movement production resulting in decrement of healthy limb performance, but also, enhanced hemiparetic performance. Two studies (Cohn, 1951; Hausmanowa-Petrusewicz, 1959) favour the compromise hypothesis.

As early as 1902 Babinski noticed that supination/pronation movement of the normal hand was disrupted by attempting to force a hemiparetic wrist to mimic the action. Cohn (1951) specifically addressed the problem of this interaction in a study of these alternating movements. He found that the rhythm and amplitude of movements on the healthy side were severely disrupted, whereas improvement in the performance of the paretic limb was very slight and in most cases frequently absent. Also, it transpired that the wrist movements appeared synchronised with the timing being determined by the slower action of the affected arm.

This was analysed in more detail by Hausmanowa-Petrusewicz (1959). She studied discrete movements, reasoning from Babinski (1902) that impairment of alternation of action need not necessarily entail impairment of more elementary actions. Her results were similar to Cohn's, but in addition and contrary to Cohn revealed that interaction also had an adverse effect upon hemiparetic limb performance.

Athough Hausmanowa-Petrusewicz aimed to study discrete actions, examination of the task employed, (repeated pressing by the hand of a rubber ball) and of her data, strongly suggests that she was in fact measuring performance on a task involving alternating movements. In keeping with Babinski's finding, Twitchell

(1951) noted the difficulty stroke patients have in changing from flexion to extension of the arm. Additionally in Experiment 3.3 it was demonstrated that alternation of synergy is a problem for the affected arm. Therefore, in order to look at individual flexion or extension movements, the tasks involved in this Chapter consist of simple, unidirectional target aiming movements.

## Experiment 4.1 - Simultaneous reaching of the two arms

### Introduction

In contrast to Experiment 3.4, this experiment involved voluntary movement of the unaffected arm instead of a isometric contraction. Subjects were required to make discrete extension or flexion movements of the arms, both bilaterally and unilaterally in the transverse plane of the shoulder. Conditions were such as to permit the highest degree of motor control the patients could elicit by allowing full visual control at all times and providing clearly defined targets. It is unclear from Cohn (1951) and Hausmanowa-Petrusewicz (1959) whether their experimental situations afforded this calibre of control. The basic question posed was, how would hemiparetic subjects spontaneously execute double arm movements and would the performance of the affected limb be influenced when the unaffected limb was moving at the same time? In addition, following from the work of Walshe (1923), Cernacek (1961), and Preilowski (1975), and the results of Experiment 3.4, which suggest bilateral interaction biases the spinal processes in favour of a muscle specific action, half of the double limb conditions entailed the use of nonhomologous muscles, whilst in the other half the same muscle groups

were employed.

## Method

### Design

The subject had to move a dowel from a starting base to a target. For each arm there were three conditions of co-ordination (unilateral-bilateral, bilateral-homologous and bilateral-nonhomologous) and two direction conditions (extension/flexion) generating eight conditions altogether in a nested design:-

Unilateral Conditions

1) RE - extension of right arm

2) RF - flexion of right arm

3) LE - extension of left arm

4) LF - flexion of left arm

Bilateral Homologous Conditions

5) RELE- right extension and left extension

6) RFLF- right flexion and left flexion

Bilateral Nonhomologous Conditions

7) RELF- right extension and left flexion

8) RFLE- right flexion and left extension

### Subjects

The subjects who took part in this experiment, had all been diagnosed as having suffered a stroke due to a CVA. Only those with no comprehensional difficulties, no perceptual dysfunction and no other motor disability, were selected. As the recording of the experiment

Table 4.1.1

| SUBJECT | AGE | POST-CVA | SEX | SP | SHOUL | ELB | WR | HAND |
|---|---|---|---|---|---|---|---|---|
| CA | 64 | 11 mths | M | N | 4 | 4 | 3+ | 3+ |
| GO | 61 | 6 mths | M | Y | 2+ | 2+ | 2 | 2 |
| MU | 32 | 27 mths | F | Y | 3+ | 2+ | 2 | 2 |
| MY | 76 | 8 mths | M | Y | 2+ | 2+ | 2 | 2 |
| SM | 45 | 4 mths | M | Y | 3 | 3 | 2 | 2 |
| TO | 69 | 4 mths | F | Y | 2+ | 3 | 2 | 2 |
| TU | 56 | 7 mths | M | N | 4 | 4 | 3 | 3 |

Columns 6-9 are MRC muscle power scalings referring only to those muscle groups necessary for arm extension. The fifth column represents a simple indication of whether (Y) or not (N) spasticity was assessed as interfering with extension.

demanded computer facilities this entailed subjects visiting the Department of Psychology. It was therefore imperative that they were able to travel and have reasonable locomotor ability. Suitable patients were drawn from the out-patient department of Astley Ainslie Hospital and from those undergoing treatment for stroke by community physiotherapists in the South Lothian District. All of the hemiparetic subjects who took part in this study are detailed in Table 4.1.1. Six of those subjects (CA, GO, MU, MY, SM, TU) took part in this experiment. All had suffered left hemisphere lesions. Normative data was provided by AL, a 63 year old woman with no history of perceptual or motor dysfunction.

Apparatus

Targets/home bases comprised 2.5cm square brass plates centred on a table 90 cms high. This height of table was chosen to bring the arm of the seated subject into the horizontal plane of the shoulder. The targets/bases were positioned such that the hands rested equidistant from the body, 24cms apart with a task distance of 24cms.

Movement was recorded by a Selspot movement monitoring system (Selcom, Sweden). The camera employed by this system consists of a position sensitive photodetector mounted in the focal plane of a Cannon ITV 50mm, 1:0.95 lens and detects the position of images of light emitting diodes (LED'S). The camera was mounted 2.13m directly above the subject and the table. The

LED'S weighed less than 2g and measured 25mm x 13mm. Six were placed on the joints of the arms. A further two were mounted on the tops of 2.5cm wooden dowels which the subject held. A ninth was fixed on the table within the field of view of the camera outwith obstruction range of the limbs. The error of measurement using this sytem is +/- 0.001% of the full width of field of the camera.

The dowels were used by the subjects to strike the targets. The dowel surfaces making contact with the bases and targets were coated by brass foil. These surfaces and the targets/bases were linked via a circuit to the LED positioned on the table. The circuit served as a twin pulse generator. When the dowels either made or broke contact with the brass plates, a series of pulses activated the LED differentially for each arm thus providing accurate timing information on the start and completion of the movements.

Fine wires connected the LED'S to a power source with all but the ninth continously emitting light. The camera was connected via a cable to the Selspot main control unit which processed the signal digitally and outputted the LED positional data along a ribbon cable to a PDP11/34 computer. Two experimenters were involved. One operated the computer, whilst the other, a qualified physiotherapist, remained with the subject for the entire course of the experiment. A buzzer served as the signal

to move, with the tone being emitted simultaneously with the commencement of sampling by the PDP11.

Procedure

The subject was seated at a table with his back straight, trunk symmetrical, hips and legs semi-flexed and feet flat on the floor. His arms rested horizontally at shoulder height with the elbow in partial flexion and forearm and wrist in the mid-position. The physiotherapist then fixed the LED'S to the subject's limbs with surgical tape as shown in Figure 4.1.1. The shoulder LED'S were placed on the superior aspect/lateral end of the acromion process. At the elbow they were positioned mid-way between the elbow skin crease and the lateral epicondyle of the humerus. A further two were fixed on the radial styloids of the wrists.

The nature of the task was explained to the subject with the instructions emphasising speed and accuracy and stressing that the dowel was not to be slid across the movement distance. In contrast to previous experiments in this thesis, the allowable margin for end point errors was relatively large: the subject was required to hit any point on the target with any part of the end of the dowel. Pilot work had indicated that if fine end point precision was required, any simultaneity between the limb movements was disrupted by switching of attention between the arms in the latter portion of the trajectories. Therefore, the experimental set-up was designed such that

Figure 4.1.1. - Showing LED's positioned on shoulder, elbow, and wrist of both arms. Subject is holding the wooden dowels with the right arm positioned for flexion and the left arm at the starting position for extension.

a normal subject could consistently hit the target within one submovement. This was the case for all the experiments in this Chapter. The nearer pair of brass plates served as starting bases for extension movements and as targets for flexion movements. Conversely, the further pair were starting points for flexion and targets for extension. Practice trials were then allowed for as long as the subject wished and only when the experimenters were satisfied that the subject fully understood the tasks was the experiment commenced.

Each of the eight conditions consisted of eight trials which were randomly presented across conditions. Before a trial was executed the physiotherapist checked that the subject's posture was erect and symmetrical. Following a verbal "Ready" signal there was a 1 to 6 second variable foreperiod before activation of the buzzer stimulus to move and simultaneous commencement of sampling.

Movements were sampled every 9.2 msec for 4.25 secs and were monitored throughout by the physiotherapist. If the movement duration exceeded the sample time, if the subject under- or over- shot the target or if he slid part of the way to it, the trial was repeated.

Rest periods were allowed at any time during the 64 trials at the request of the subject or if the

physiotherapist judged him to be becoming fatigued.

Data Analysis

Of principal interest is a comparison of movement trajectories across the experimental conditions. Figures 4.1.2A and 4.1.3A show the change in position of the left (unaffected) and right (affected) arms over the course of a typical single extension movement. As these actions were performed primarily within the subject's transverse plane, along the sagittal axis (the Y-axis of the figures), analysis is restricted to that direction and to the path of the limb extremity. This movement path was obtained from the change in wrist position over the trial. Position curves were then differentiated to obtain velocity profiles. Figures 4.1.2B and 4.1.3B illustrate the wrist velocity profiles of Figures 4.1.2A and 4.1.3A respectively.

Crucial to the analysis was the identification of units of movement, or submovements, as measures of smoothness of trajectory. The system adopted to do this was essentially the same as in Chapter III. However because the movements in this experiment were performed in three dimensions, instead of being constrained to one, it was difficult to separate submovements composing the trajectory from postural adjustments before lift-off and after touch down. The overall number of submovements was calculated with the aid of lift-off and touch-down times of the dowel, as determined by the contact plates. Using these values, the start of the movement was defined

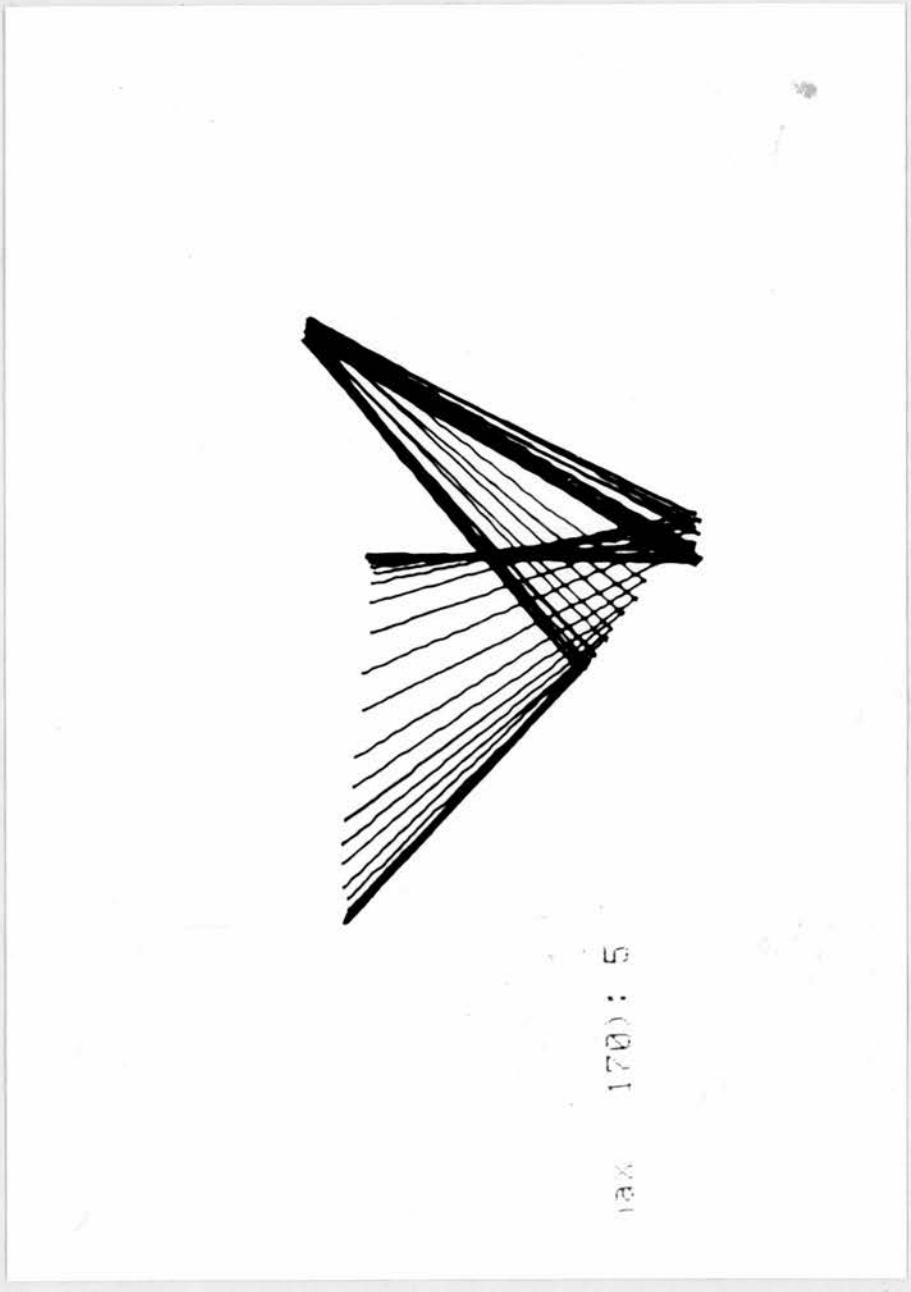



Figure 4.1.2A - Left (unaffected) arm. "Multiple exposure" over a trial of processed LED signals with the points joined in a stick arm.

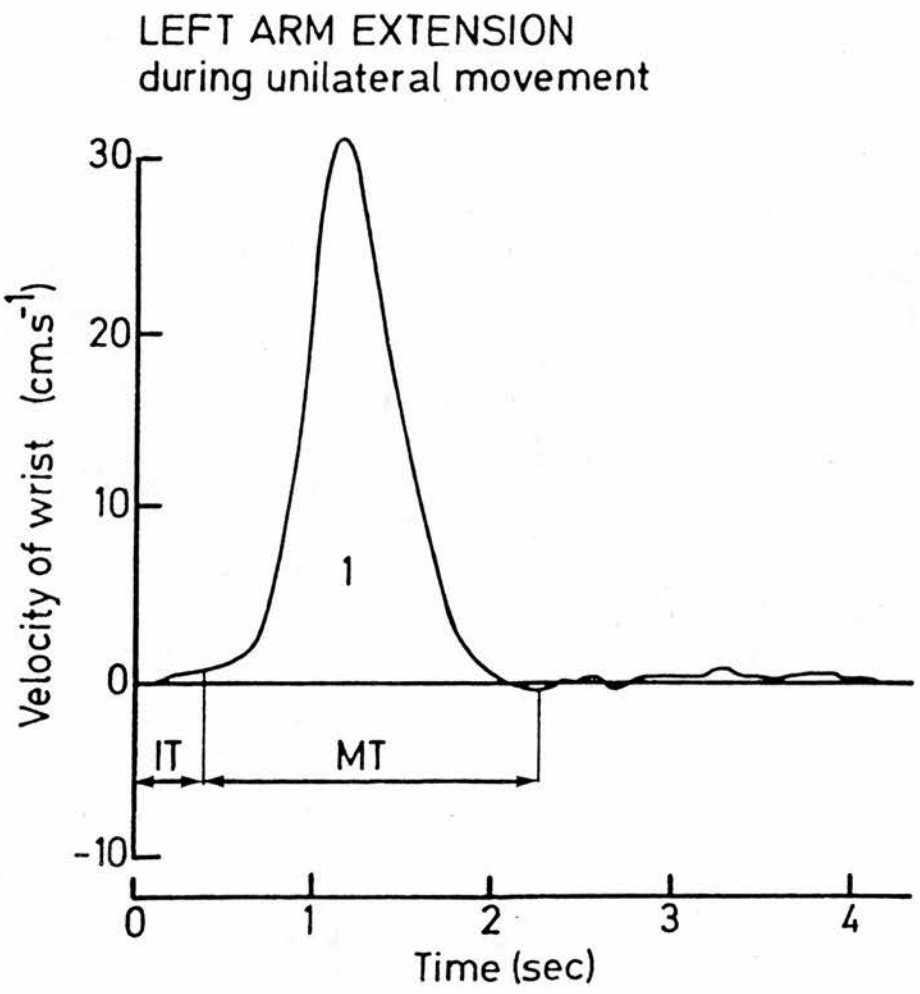



Figure 4.1.2B Velocity profile of the wrist from Figure 4.1.2A.

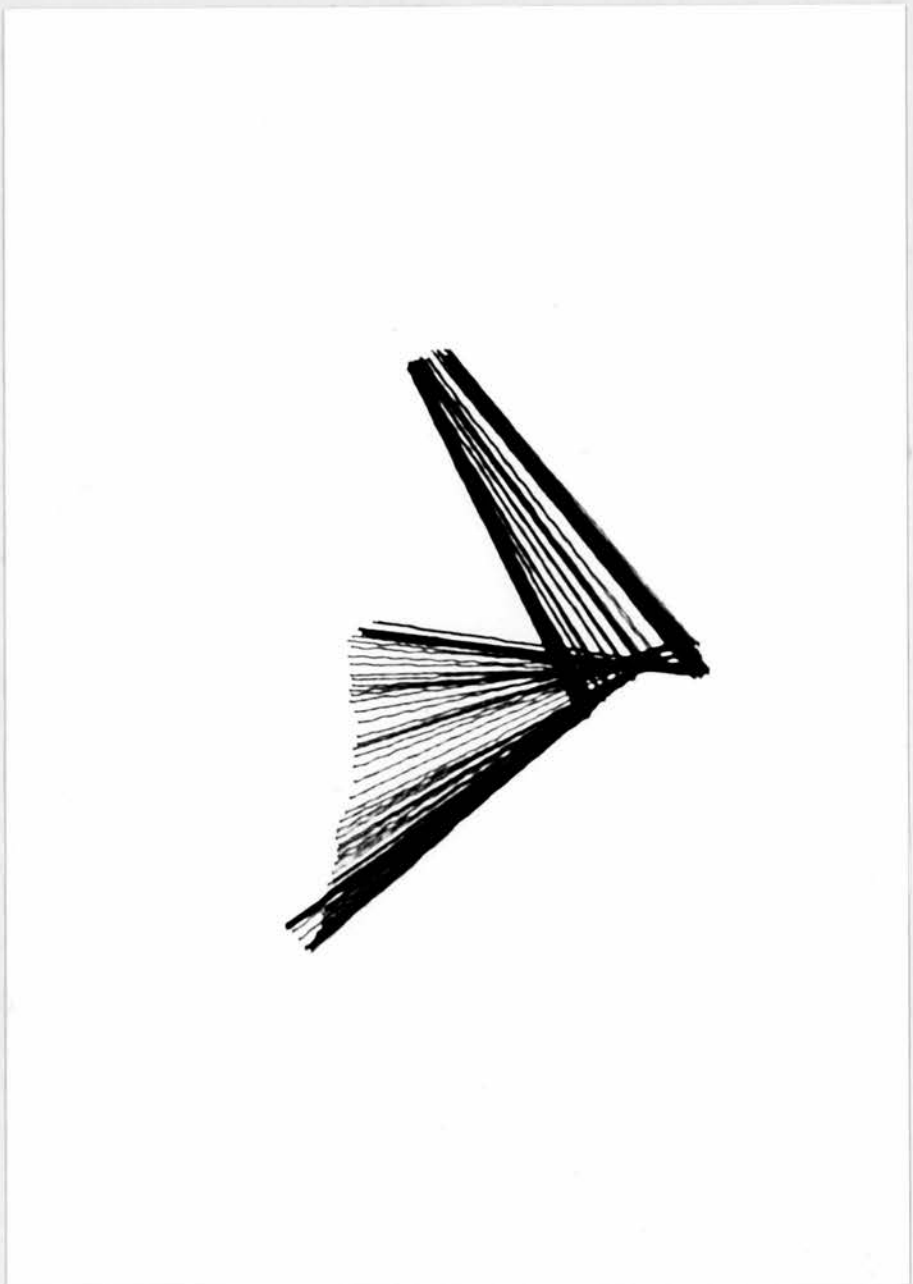


Figure 4.1.3A - As Figure 4.1.2A for the right (affected) arm.

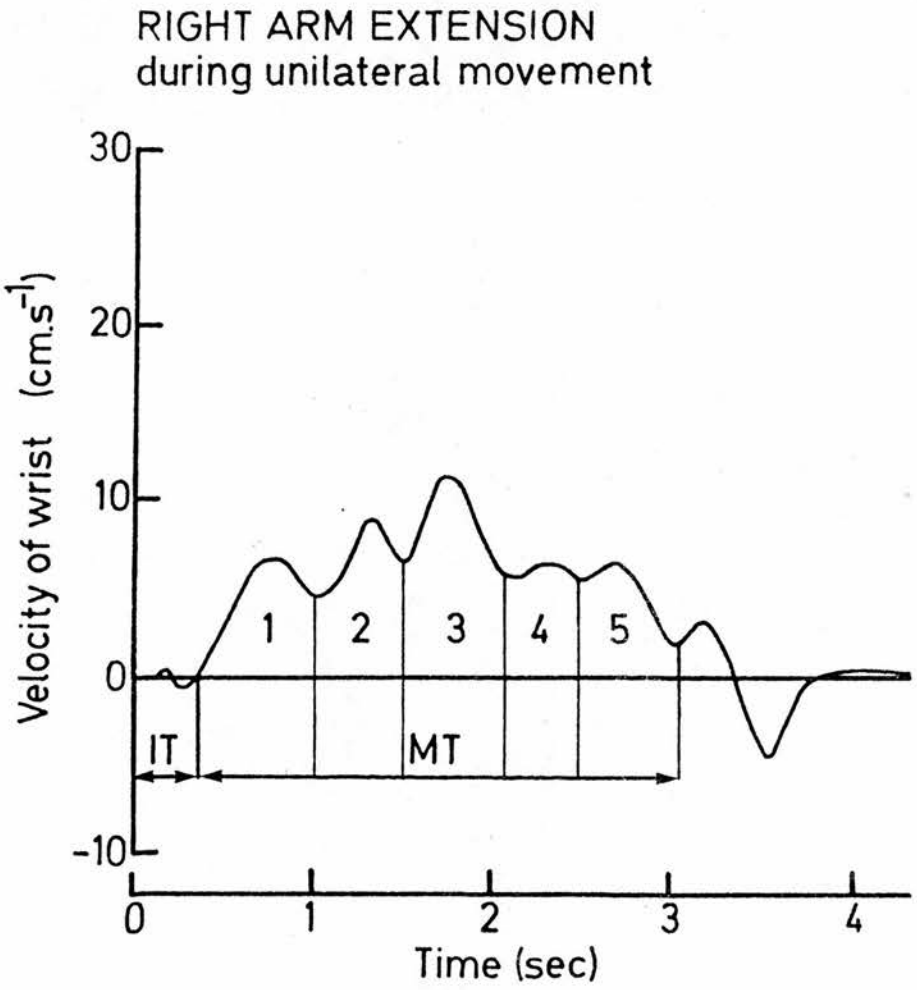



Figure 4.1.3B Velocity profile of the wrist from Figure 4.1.3A.

as the start of the submovement encompassing the lift-off value. Completion of movement was recognised by the end of the submovement during which touch-down occurred.

Figure 4.1.3B shows a velocity profile composed of five submovements with each visible as peaks on the record. There are, however, more than five peaks altogether, but since this study focuses on the trajectory of limb translation, the other submovements present are identified as being postural adjustments of the limb prior to and after execution of the task movements. These are, therefore, rejected in calculation of the number of submovements.

Figures 4.1.2B and 4.1.3B also demonstrate how the other variables under consideration here were obtained. Initiation time (IT) is given by the start time of the movement, the start signal being at time zero. Movement time (MT) is the difference between start and finish times.

## Results

### Simultaneity of action

Means and standard deviations of initiation times (IT) for each condition are listed for each subject in Table 4.1.2. Individual 3-way repeated measures ANOVA's, 2x2x3, (Arm (A) x Direction (D) x Co-ordination (C)) were performed on each subject's data. These revealed that in all subjects, including the control subject, AL, there was no main effect of Arm. Over all subjects, there were only three interactions involving this factor (MU: AxC, $F=4.43$ $dF=1,7$ $p< 0.05$; MY: AxD, $F=8.68$ $dF=1,7$ $p < 0.05$; SM: AxD, $F=24.50$ $dF=1,7$ $p< 0.01$) and when individual comparisons between these were done using Scheffe tests it was found that the significant effect is due to differential effects of the levels of the other factors. Therefore, in bilateral movements there are no significant differences between the arms as regards time to prepare and commence activity. The lack of AxC interactions means that when moving unilaterally the unaffected arm is no quicker off the mark than the affected.

When movement time (MT) means (Table 4.1.3) are analysed, the control subject AL shows no main effect of arm, nor any significant interaction involving it, but all the hemiparetic subjects show a main effect (GO: A, $F=171.74$, $dF=1,7$, $p<0.001$; TU: A, $F = 35.56$, $dF = 1,7$, $p<0.001$; CA: A, $F = 5.86$, $dF = 1,7$, $p<0.05$; MU: A, $F =$

Table 4.1.2
Mean (SD) Initiation Time (msec)

| Movement Condition | Subjects MU | SM | MY | GO |
|---|---|---|---|---|
| LE | 234 | 407 | 266 | 382 |
| | (019) | (125) | (065) | (135) |
| LE(RE) | 256 | 554 | 314 | 513 |
| | (074) | (244) | (093) | (087) |
| LE(RF) | 230 | 537 | 343 | 545 |
| -------- | (039) | (243) | (181) | (245) |
| LF | 266 | 454 | 487 | 422 |
| | (069) | (249) | (183) | (201) |
| LF(RF) | 241 | 459 | 413 | 313 |
| | (037) | (143) | (181) | (095) |
| LF(RE) | 220 | 646 | 268 | 435 |
| -------- | (031) | (233) | (073) | (171) |
| RE | 219 | 600 | 372 | 600 |
| | (037) | (159) | (147) | (225) |
| RE(LE) | 239 | 800 | 417 | 601 |
| | (034) | (280) | (132) | (247) |
| RE(LF) | 291 | 658 | 388 | 670 |
| -------- | (096) | (108) | (147) | (263) |
| RF | 219 | 424 | 230 | 499 |
| | (021) | (229) | (043) | (170) |
| RF(LF) | 243 | 407 | 341 | 369 |
| | (048) | (163) | (114) | (127) |
| RF(LE) | 260 | 439 | 332 | 473 |
| | (074) | (172) | (211) | (176) |

Table 4.1.2
Mean (SD) Initiation Time (msec)

| Movement Condition | Subjects CA | TU | AL |
|---|---|---|---|
| LE | 252 | 290 | 203 |
| | (061) | (080) | (026) |
| LE(RE) | 248 | 259 | 250 |
| | (043) | (068) | (058) |
| LE(RF) | 301 | 391 | 264 |
| | (124) | (148) | (068) |
| LF | 269 | 303 | 205 |
| | (069) | (130) | (033) |
| LF(RF) | 291 | 283 | 240 |
| | (130) | (152) | (072) |
| LF(RE) | 337 | 380 | 233 |
| | (239) | (361) | (041) |
| RE | 292 | 293 | 201 |
| | (080) | (122) | (034) |
| RE(LE) | 268 | 347 | 231 |
| | (049) | (194) | (041) |
| RE(LF) | 290 | 418 | 225 |
| | (144) | (368) | (035) |
| RF | 289 | 306 | 249 |
| | (056) | (137) | (068) |
| RF(LF) | 308 | 266 | 240 |
| | (118) | (134) | (037) |
| RF(LE) | 322 | 315 | 247 |
| | (150) | (116) | (068) |

Table 4.1.3

Mean (SD) Movement Time (sec)

| Movement Condition | Subjects MU | SM | MY | GO |
|---|---|---|---|---|
| LE | 0.904 | 1.689 | 1.348 | 1.256 |
| | (0.108) | (0.420) | (0.182) | (0.365) |
| LE(RE) | 1.109 | 1.887 | 1.646 | 1.483 |
| | (0.253) | (0.397) | (0.222) | (0.536) |
| LE(RF) | 0.920 | 2.311 | 1.323 | 1.386 |
| | (0.128) | (0.184) | (0.248) | (0.383) |
| LF | 0.884 | 1.648 | 0.995 | 1.070 |
| | (0.091) | (0.128) | (0.164) | (0.180) |
| LF(RF) | 0.948 | 1.999 | 1.387 | 1.453 |
| | (0.188) | (0.393) | (0.308) | (0.508) |
| LF(RE) | 0.859 | 1.890 | 1.066 | 1.437 |
| | (0.143) | (0.392) | (0.197) | (0.440) |
| RE | 1.217 | 2.365 | 2.096 | 2.065 |
| | (0.205) | (0.396) | (0.609) | (0.676) |
| RE(LE) | 1.262 | 2.853 | 2.090 | 2.103 |
| | (0.124) | (0.422) | (0.384) | (0.272) |
| RE(LF) | 1.213 | 3.184 | 2.318 | 2.374 |
| | (0.432) | (0.476) | (0.752) | (0.548) |
| RF | 1.290 | 1.868 | 1.500 | 1.827 |
| | (0.317) | (0.332) | (0.330) | (0.471) |
| RF(LF) | 1.819 | 2.198 | 1.585 | 1.974 |
| | (0.408) | (0.209) | (0.507) | (0.357) |
| RF(LE) | 1.672 | 2.389 | 1.754 | 2.325 |
| | (0.201) | (0.413) | (0.515) | (0.444) |

Table 4.1.3
Mean (SD) Movement Time (sec)

| Movement Condition | Subjects CA | TU | AL |
|---|---|---|---|
| LE | 0.840 | 0.963 | 0.334 |
| | (0.106) | (0.135) | (0.052) |
| LE(RE) | 1.087 | 0.987 | 0.375 |
| | (0.135) | (0.091) | (0.062) |
| LE(RF) | 0.982 | 1.104 | 0.461 |
| -------- | (0.227) | (0.059) | (0.079) |
| LF | 0.989 | 0.995 | 0.404 |
| | (0.348) | (0.146) | (0.067) |
| LF(RF) | 0.873 | 1.069 | 0.377 |
| | (0.190) | (0.228) | (0.037) |
| LF(RE) | 0.812 | 1.157 | 0.439 |
| -------- | (0.116) | (0.190) | (0.081) |
| RE | 0.935 | 1.171 | 0.342 |
| | (0.149) | (0.187) | (0.039) |
| RE(LE) | 1.114 | 1.233 | 0.376 |
| | (0.122) | (0.676) | (0.055) |
| RE(LF) | 1.107 | 1.210 | 0.474 |
| -------- | (0.183) | (0.152) | (0.094) |
| RF | 0.913 | 1.135 | 0.344 |
| | (0.176) | (0.230) | (0.046) |
| RF(LF) | 0.889 | 1.039 | 0.394 |
| | (0.196) | (0.140) | (0.081) |
| RF(LE) | 0.973 | 1.316 | 0.431 |
| | (0.208) | (0.278) | (0.088) |

Table 4.1.4

Correlations between the arms Sig: > .707

| SUBJECT | CONDITION | IT | TPV | FT |
|---|---|---|---|---|
| MU | RELE | +.300 | +.056 | +.290 |
| | RFLE | +.381 | +.288 | +.513 |
| | RFLF | +.522 | +.234 | +.246 |
| | RELF | +.561 | +.127 | +.112 |
| SM | RELE | +.560 | +.871 | +.230 |
| | RFLE | +.026 | +.205 | +.531 |
| | RFLF | +.652 | +.940 | +.273 |
| | RELF | +.403 | +.214 | +.260 |
| MY | RELE | +.491 | +.464 | -.425 |
| | RFLE | +.927 | +.821 | +.779 |
| | RFLF | +.616 | +.977 | -.175 |
| | RELF | -.126 | -.090 | -.274 |
| GO | RELE | +.354 | +.287 | -.050 |
| | RFLE | +.576 | +.469 | -.577 |
| | RFLF | +.119 | +.086 | +.595 |
| | RELF | +.156 | +.175 | +.122 |
| CA | RELE | +.607 | +.723 | +.471 |
| | RFLE | +.649 | +.743 | +.506 |
| | RFLF | +.968 | +.982 | +.762 |
| | RELF | +.986 | +.945 | +.660 |
| TU | RELE | +.124 | +.359 | +.547 |
| | RFLE | +.858 | +.725 | +.851 |
| | RFLF | +.980 | +.927 | +.861 |
| | RELF | +.981 | +.992 | +.846 |
| AL | RELE | +.993 | +.325 | +.956 |
| | RFLE | +.812 | +.952 | +.864 |
| | RFLF | -.089 | +.884 | +.938 |
| | RELF | +.908 | +.665 | +.874 |

66.07, dF = 1,7, p< 0.001; MY: A, F = 86.22, dF = 1.22, dF = 1,7, p< 0.001; SM: A, F = 43.91, dF = 1,7, p<0.001). Three of these subjects also show interaction effects for AxD (MU: AxD, F = 18.95, dF = 1,7, p<0.01; MY: AxD, F = 7.53, dF = 1,7, p<0.05; SM: AxD, F = 8.94, dF = 1,7, p<0.005) which can be attributed to extension / flexion differences in the affected arm.

Although three of the six hemiparetic subjects have significant effects for Co-ordination (GO: C, F = 3.69, dF = 2,14, p<0.05; MU: C, F = 20.48, dF = 2,14, p<0.001; SM C, F = 13.29, dF = 2,14, p<0.001) and over all the subjects there is a trend, in both arms, for bilateral movements to take longer to execute than unilateral movements, it cannot be stated that there is a parity of movement time. Therefore, in general, regardless of condition, the action of the affected arm is slower than the unaffected.

Therefore, the picture emerging is that, although in hemiparetic bilateral movements the two arms start moving at around the same time, the affected arm takes longer to complete the task. In order to discover any timing relationships between the arms, correlations (Pearson's r) were done for IT, time to peak velocity and time to completion of action (Table 4.1.4). Only two of the stroke patients (CA and MU) show any substantial timing relationship, indicating that although the hemiparetic

subjects don't plan and execute bilateral movements independently, there is little evidence of temporal coupling of the limbs at key points in the trajectories.

Smoothness of Trajectory

Figure 4.1.4 shows mean number of submovements present in the movement path of each limb across all the conditions for the hemiparetic subjects as obtained from Table 4.1.5. Control subject AL's data is not presented here as she consistently performed the tasks within one submovement. As above, 3-way ANOVAs were conducted for each subject. TU and CA show almost perfect performance on this measure, in both arms and analysis revealed no significant main effects or interactions. Therefore, discussion in this section concerns the results of the other four hemiparetic subjects.

Considering extension movements, as Figure 4.1.4 shows, the right arm movements contain more submovements than the left. This is reflected in a main effect of Arm in the four subjects (MU: A, $F = 148.9$, $dF = 1,7$, $p < 0.001$; MY: A, $F = 214.5$, $dF = 1,7$, $p<0.001$; SM: A, $F = 326.8$, $dF = 1,7$, $p<0.001$; GO: A, $F = 46.9$, $dF = 1,7$, $p<0.01$). However, as the graph also shows, the magnitude of difference between the means across the arms varies according to the level of Co-ordination, with a tendency for double homologous movements to have the least magnitude of difference. But when comparisons across

Table 4.1.5
Mean (SD) Number of Submovements

| Movement Condition | Subjects MU | SM | MY | GO |
|---|---|---|---|---|
| LE | 1.00 | 1.13 | 1.13 | 1.13 |
| | (0.00) | (0.38) | (0.35) | (0.35) |
| LE(RE) | 1.25 | 2.13 | 1.38 | 1.88 |
| | (0.46) | (1.36) | (0.52) | (0.99) |
| LE(RF) | 1.38 | 1.88 | 1.13 | 1.50 |
| | (0.74) | (0.84) | (0.35) | (0.76) |
| LF | 1.00 | 1.38 | 1.13 | 1.13 |
| | (0.00) | (0.52) | (0.35) | (0.35) |
| LF(RF) | 1.13 | 1.25 | 1.25 | 1.63 |
| | (0.35) | (0.46) | (0.46) | (0.74) |
| LF(RE) | 1.13 | 1.38 | 1.00 | 1.75 |
| | (0.35) | (1.06) | (0.00) | (0.71) |
| RE | 2.88 | 3.88 | 3.38 | 3.00 |
| | (0.35) | (0.99) | (0.74) | (0.76) |
| RE(LE) | 2.25 | 3.88 | 2.75 | 2.25 |
| | (0.46) | (0.64) | (0.71) | (0.44) |
| RE(LF) | 3.13 | 5.13 | 4.25 | 3.50 |
| | (0.64) | (0.99) | (1.49) | (1.07) |
| RF | 2.38 | 1.75 | 2.13 | 2.63 |
| | (0.92) | (0.71) | (0.99) | (0.92) |
| RF(LF) | 3.00 | 2.13 | 3.13 | 2.13 |
| | (1.07) | (0.99) | (2.17) | (0.99) |
| RF(LE) | 3.50 | 2.00 | 2.25 | 3.63 |
| | (0.93) | (0.93) | (1.28) | (1.30) |

Table 4.1.5
Mean (SD) Number of Submovements

| Movement Condition | Subjects CA | TU |
|---|---|---|
| LE | 1.00 | 1.00 |
| | (0.00) | (0.00) |
| LE(RE) | 1.00 | 1.00 |
| | (0.00) | (0.00) |
| LE(RF) | 1.13 | 1.00 |
| | (0.35) | (0.00) |
| LF | 1.13 | 1.00 |
| | (0.35) | (0.00) |
| LF(RF) | 1.00 | 1.00 |
| | (0.00) | (0.00) |
| LF(RE) | 1.00 | 1.00 |
| | (0.00) | (0.00) |
| RE | 1.25 | 1.25 |
| | (0.46) | (0.46) |
| RE(LE) | 1.13 | 1.25 |
| | (0.35) | (0.35) |
| RE(LF) | 1.00 | 1.25 |
| | (0.00) | (0.00) |
| RF | 1.13 | 1.13 |
| | (0.35) | (0.35) |
| RF(LF) | 1.13 | 1.00 |
| | (0.35) | (0.00) |
| RF(LE) | 1.13 | 1.38 |
| | (0.35) | (0.35) |

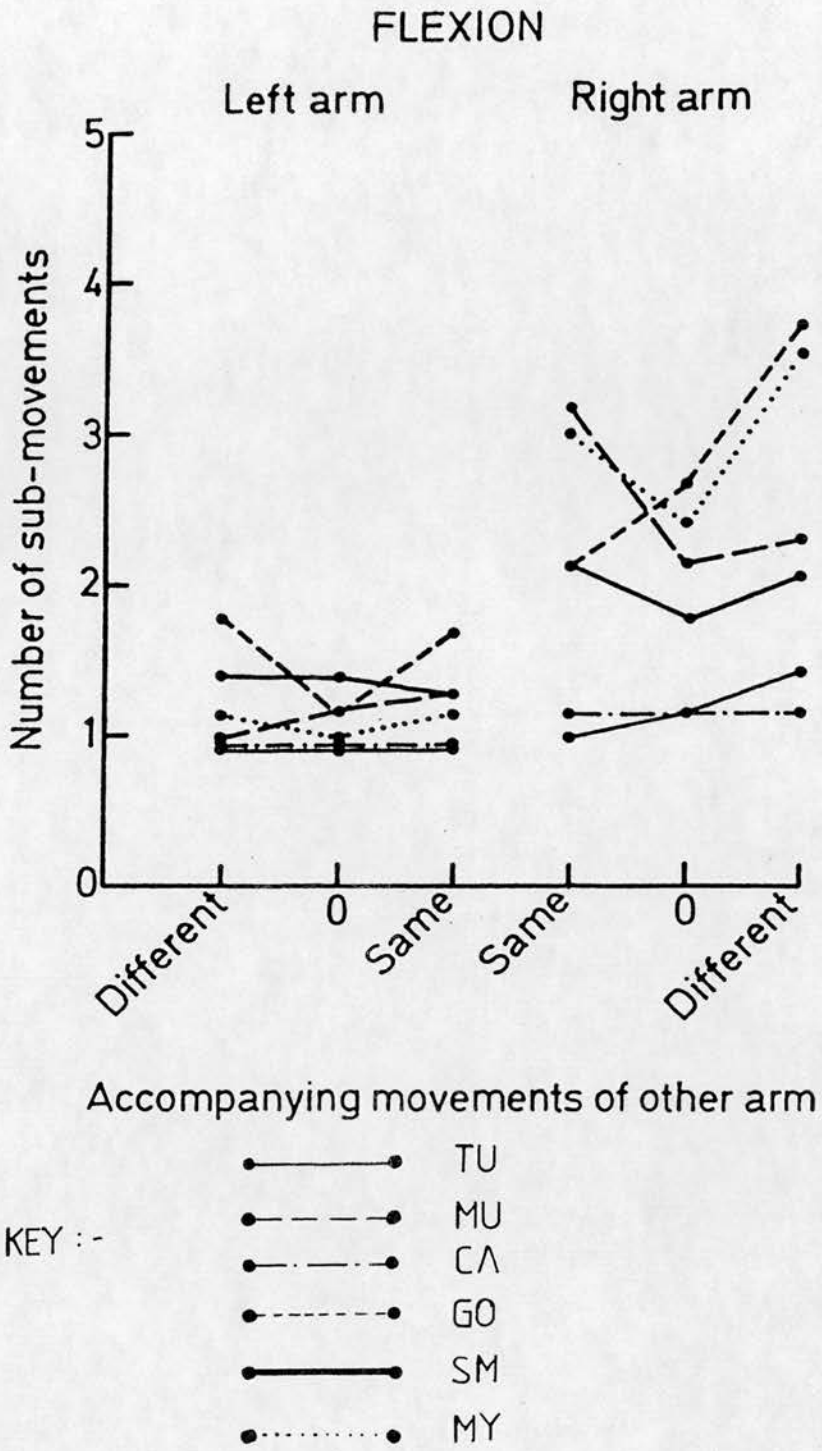
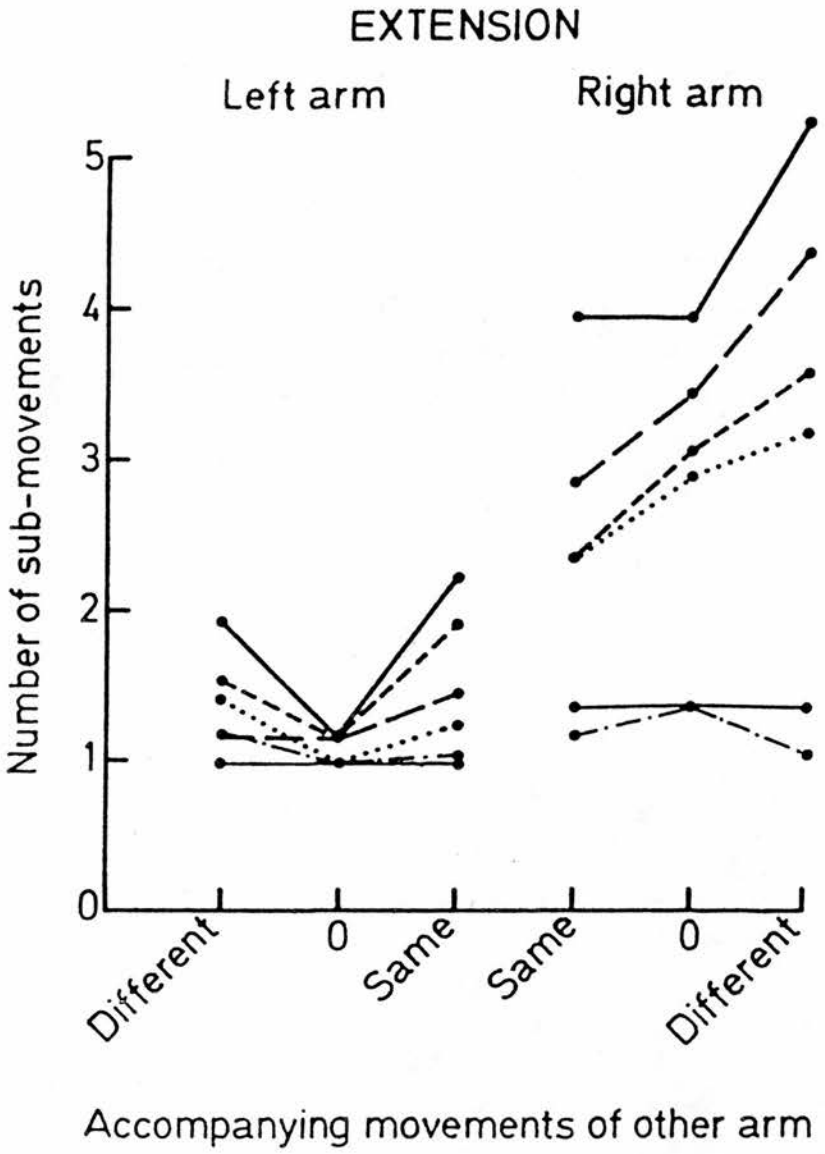



Figure 4.1.4 Mean number of submovements.

Different : Extension
Same : Flexion
0 : No movement




Different : Flexion
Same : Extension
0 : No movement

Figure 4.1.4 Mean number of submovements.

arms between individual means were done using Scheffe tests, all but subject (GO) showed significant differences.

Two points of note emerge from this. Firstly, there is no detrimental effect on unaffected limb performance as would be predicted by the compromise hypothesis does not happen: the results are more in keeping with the consensus hypothesis. Secondly, the consensus hypothesis appears only to apply to homologous double movement and not non-homologous movement. These conclusions can be substantiated by an examination of the effects of Co-ordination on extension.

Co-ordination is a main effect in three out of the four subjects (MU: C, $F = 9.0$, $dF = 2,14$, $p<0.01$; SM: C, $F = 6.2$, $dF = 2,14$, $p<0.05$; GO: C, $F = 7.5$, $dF = 2,14$, $p<0.01$). Individual comparisons using Scheffe tests show that, with the exception of subject SM, unilateral right extension contains significantly more submovements than right extension, when coupled with left extension, and in all three subjects right extension at the same time as left flexion is significantly more discontinuous than in the double homologous instance. In movements of the left arm, GO and SM show left extension coupled with right extension to have significantly more submovements than left extension alone. Therefore, the hypothesis which best fits the extension results is the

consensus hypothesis.

The plot of number of submovements for flexion does not show as clear a picture and the presence of significant interactions involving Direction and Co-ordination, in two of the four subjects (MU: AxDxC, $F = 4.7$, $dF = 2,14$, $p<0.05$; SM DxC, $F = 4.9$, $dF = 2,14$, $p<0.05$) underlines this difference. Only subject GO shows the pattern of results across the levels of Co-ordination found above for extension. Contrary to this, the other three subjects demonstrate a rise in number of submovements going from unilateral to bilateral, but this is only significant in Scheffe comparisons for MU.

Extension/Flexion Differences

Comparing unilateral movements, there are significant differences ($p<0.05$) for MU, SM and MY and the presence of A x D interactions in two of the subjects (MY: A xD, $F= 5.9$, $dF = 1,7$, $p<0.05$; SM: AxD, $F = 52.4$, $dF = 1,7$, $p<0.01$) provide some support for flexion being accomplished in less submovements than extension. It seems likely however, that despite the presence of only one Direction x Co-ordination interaction (SM: DxC, $F = 4.9$, $dF = 2,14$, $p<0.05$) differences between hemiparetic extension and flexion is confounded by the levels of Co-ordination. Subject MU demonstrates this with right extension having significantly more submovements than

right flexion in the unilateral condition, but significantly less in the bilateral homologous condition.

There is a suggestion in the results that flexion of the affected arm would take less time to initiate than extension. The presence of the two significant Arm x Direction interactions already noted, lends some support to this argument which can be seen as the trend across the means of all hemiparetic subjects with the exception of CA.

Main effects of Direction on MT are present in three of the hemiparetic subjects (MU: D, $F = 8.70$, $dF = 1,7$, $p<0.05$; MY: D, $F = 40.56$, $dF = 1,7$, $p<0.001$; SM: D, $F = 80.58$, $dF = 1,7$, $p<0.001$) and these subjects also show Arm x Direction interactions (MU: AxD, $F= 18.95$, $dF = 1,7$, $p<0.01$; MY: AxD, $F = 7.53$, $dF = 1,7$, $p<0.05$; SM: AxD, $F = 8.94$, $dF = 1,7$, $p<0.05$) which in all but one case (MU) indicate flexion of the affected arm to be overtly executed more briskly than extension.

It is difficult to see any links between the pattern of results for number of submovements and those for the variation of LT across conditions. In particular, the consensus effect for double extension movements and its opposite effect in the non homologous instance cannot be explained by interaction in the preparatory stages of action as there are no main effects of Co-ordination on

IT in the four subjects and only one Arm Co-ordination interaction (MU: AxC, F= 4.43, dF = 2,14, $p<0.05$). Although, MT has main effects of Co-ordination in three out of the four subjects (MU: F = 20.48, dF = 2,14, $p<0.001$; SM: C, F = 13.29, dF = 2,14, $p<0.001$; GO: C, F = 3.69, dF = 2,14, $p<0.05$) and an interaction with Arm in one, this is, as suggested earlier, only indicative of a general rise in MT going from unilateral to bilateral. Notably, subject AL shows a significant main effect of Co-ordination (AL: C, F = 11.05, dF = 2,14, $p< 0.01$) on MT, with bilateral movements taking longer than unilateral, especially non-homologous double movements, even although she consistently accomplished the trials within one submovement.

Discussion

The data supports the idea that extension movements of the affected arm are facilitated by simultaneous homologous activity in the healthy limb. Moreover, although bilateral activity induces slight deleterious effects upon unaffected arm activity, in sharp contrast to the findings of Cohn (1951) and Hausmanowa-Petrusewicz (1959), this detriment does not result in a level of performance matching that of the affected arm. This argues strongly against the compromise model.

The reason for this contrast in findings is probably due to two major procedural differences between this study and the other two cited. As previously stated it is likely that examination of movement in both Cohn (1951) and Hausmanowa-Petrusewicz (1959) was confounded by the problem stroke patients have in alternating between different muscle synergies. Of equal and perhaps even greater importance, is the fact that in both of these studies the experimental actions were non-goal directed. All normal human activity is goal directed. In fact in cases where it is purposeless, as in athetoid movements, it is diagnosed as pathological. Therefore, in attempting to understand movement dysfunction it must be studied within a functional setting. The procedures of Cohn (1951) and Hausmanowa-Petrusewicz (1959), respectively, involved supination / pronation of the wrist and the pressing of a ball by the fingers, but in

both cases the subjects were not advised on how much movement in the required direction(s) should be made, i.e.they were given no target to aim for, other than mere production of movement. Without an obvious goal and therefore the necessity of planning and executing action with respect to it, it is highly likely that there was a bias towards the worst level of performance as there was no reference against which to gauge it. The provision of a definitive target for action bears strongly on the explanation of the consensus effect about to be presented below.

Cohn (1951) demonstrated that when the affected limb is passively moved no detriment in healthy limb performance ensues. Thus interaction effects in bilateral activity are due to descending motor influences and not propriospinal reflexes. Since not all the corticospinal fibres decussate and the subcortical descending systems have a bilateral as well as unilateral distribution, there exists the possibility of ipsilateral innervation of the affected side by the intact hemisphere. This, with the possibility of interaction between contralateral processes at the cortical, subcortical, and spinal levels provides a structural substrate for communication between the two halves of the body. Preilowski's (1975) model of bilateral activity focuses upon interaction between ipsilateral and contralateral innervation in movement production and can

explain the results obtained here of coupling affected arm extension with homologous and non-homologous movements of the other arm. The model, which is illustrated in Figure 4.1.5, was formulated following observed differences by Cohen (1970) and Preilowski (1972) between symmetrical and asymmetrical bilateral performance in normals and commissurotomy patients respectively. Asymmetric performance demonstrated interference effects, not found in the symmetric case, which were attributable to the asymmetric condition causing a conflict between the differing ipsilateral and contralateral commands. In the symmetric condition, no such conflict is present because the ipsilateral and contralateral commands both specify the same goal.

Martenuik and McKenzie's (1980) results and subsequent specification of the model to explain facilitatory and inhibitory effects in asymmetric bilateral action, fit well to the data obtained in this experiment (Figure 4.1.6). When extension of the affected arm is paired with flexion of the unaffected arm the contralateral efference specifies extension, while the ipsilateral dictates flexion (bottom half of the model). Since the nature of the lesion caused by stroke cannot be functionally defined with any certainty at the subcortical level, discussion here focuses upon bilateral interaction in the

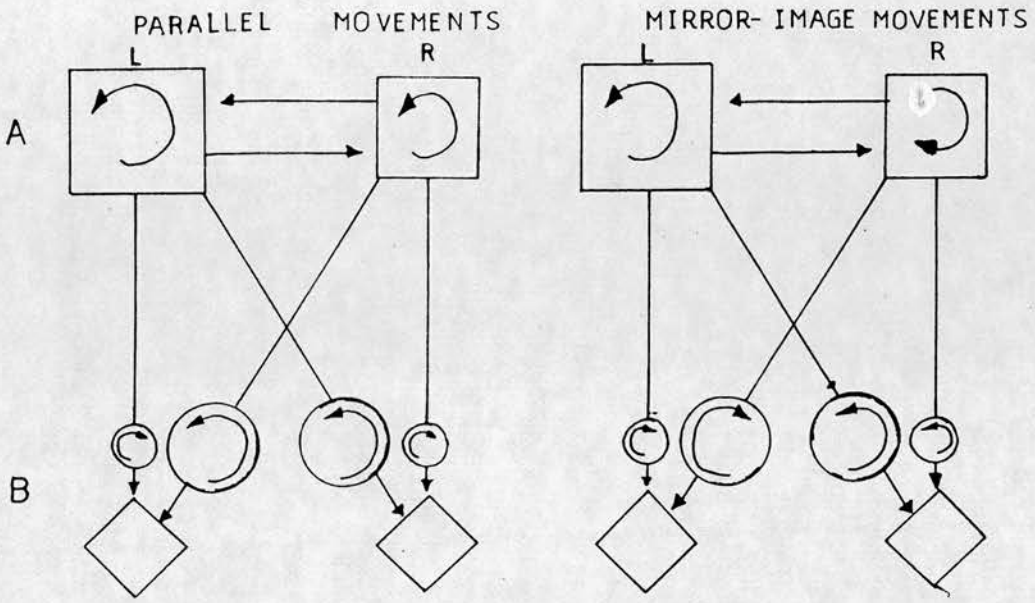



Figure 4.1.5. - Prelowski's model of bilateral interaction. (1975)
Derived from handle turning experiments comparing asymmetric ( ) with symmetric ( ) circular movements. A represents the cortical level. The circles indicate the type of motor signal transmitted to lower motor centres represented as B. The figure illustrates how the influence of ipsilateral control interacts with contralateral signals from the opposite hemisphere.

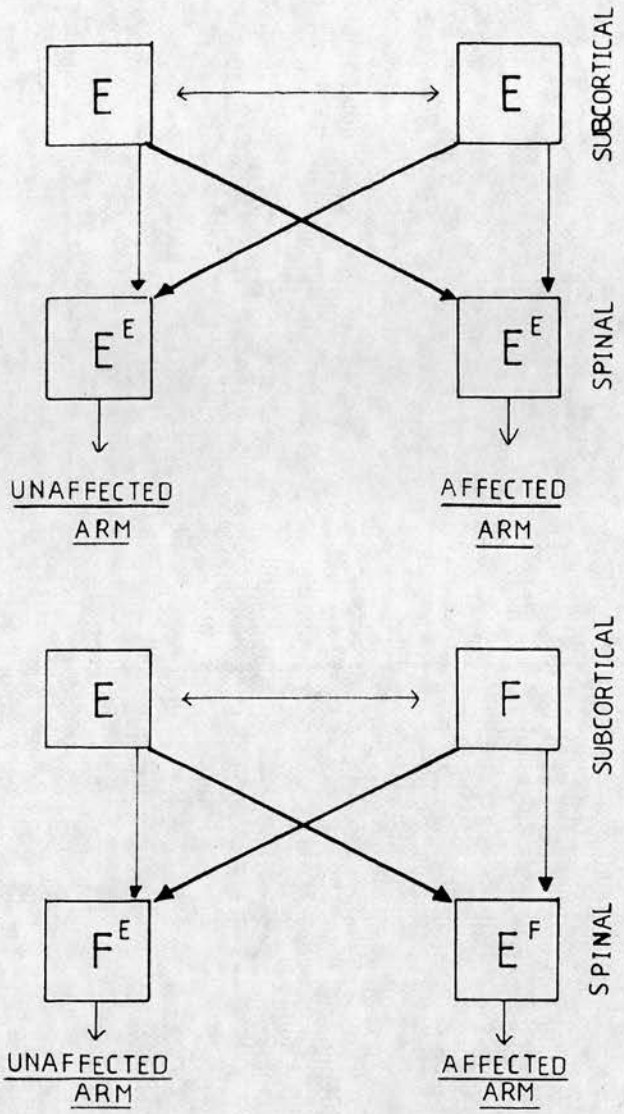



Figure 4.1.6. - Adapted from Martenuik and McKenzie's (1980) model of bilateral interaction.

Indicates how interaction between hemispheric specification of extension (E) and flexion (F) may occur at subcortical and spinal levels. Heavy arrows with shaded point s represent contralateral projection. Lighter arrows with shaded points represent ipsilateral projection.

internuncial region of the spinal cord (Brodal, 1962; Lawrence and Kuypers, 1968 (a)). As argued in previous Chapters, the principal neurological problem facing stroke rehabilitation appears to be abnormal supraspinal impingement upon the Ia interneurons mediating reciprocal innervation (Miller and Hammond, 1982). Given that for the affected arm the activity of these bodies may bias flexor activity around the elbow, even within extension movements, extension of the affected arm is interrupted by this tendency for co-contraction. This is reflected in the larger number of submovements in right extension compared to left extension or right flexion. However, when right extension is coupled with left extension (top half of the model), the additional ipsilateral bias towards extension could facilitate that movement. This indeed appears to be what happens, as the data shows a trend for the number of submovements to go down when unilateral right extension is compared with right extension in a bilateral homologous setting. As the model would predict, when left flexion is simultaneous with right extension, the number of submovements rises since the contralateral flexor bias is reinforced by an additional ipsilateral bias towards flexion which increases the co-contraction problem in extension and thus results in more interruption to the action.

## Experiment 4.2 - The importance of visual monitoring in simultaneous reaching

### Introduction

The principal question to be addressed in this experiment is to what extent the consensus effect demonstrated in Experiment 4.1 is dependent upon continuous visual monitoring. Given the results of the previous chapter it might be expected that any control process underlying hemiparetic performance will be heavily dependent on visual guidance. However there exists the possibility that the consensus effect is simply a result of uncontrolled efferent overflow. If that were the case then the effect should still be apparent when vision of both arms is withdrawn. In addition, selective removal of vision (of one arm only) should reveal more about the means by which vision could be aiding performance. For example it would be important for both arms to be visible if the effect is dependent on visually based tracking of the unaffected limb by the affected.

## Method

### Design

All the movements in this experiment were of extension. There were two movement conditions (unilateral/bilateral) mixed, where appropriate, with four viewing conditions (full vision(V)/no vision(NV)/vision of right arm only(VR)/vision of left arm only(VL)) giving eight conditions in a nested design:-

Unilateral Conditions

1) REV - extension of right arm with visual inspection permitted throughout.

2) LEV - as (1) for left arm.

3) RENV - extension of right arm with eyes closed.

4) LENV - as (3) for left arm.

Bilateral Conditions

5) RELEV - movement of both arms with vision permitted throughout.

6) RELENV - movement of both arms with eyes closed.

7) RELEVR - movement of both arms with sight of right arm only.

8) RELEVL - movement of both arms with sight of left arm only.

### Subjects

Four of the hemiparetic subjects who took part in Experiment 4.1 also participated in this experiment. They were: MU, SM, MY and TU. A fifth stroke patient,

TO, was also run. In common with the others TO had suffered a left CVA. The normal subject in this experiment was MA, a 26 year old right handed male with no history of perceptual or motor disorder.

Apparatus

The same apparatus was used as in Experiment 4.1. However in addition a screen (55cms square) was available for occluding view of one arm whilst retaining sight of the other, in bilateral movement trials with differential viewing conditions.

Procedure

Positioning of the subject and placement of the LED's was exactly as in Experiment 4.1. Again there were eight randomly presented trials for each condition. Practice, operating, and checking routines were similar with the only difference being in the practice and execution of 'NV, 'VR and 'VL conditions.

In the previous Chapters a consistent finding was that the affected arm undershot the target. Since interest in this Chapter was in the structure of the gross trajectory of movement and not accuracy, the effective target size was relatively large. However, without sight of performance it was still possible that the affected arm might undershoot even an effectively large target and confound any later interpretation of symmetrical interaction. Therefore during practice trials involving loss of vision, an attempt was made to

train the subject to hit the target by giving verbal KR. This procedure was largely successful in that only around 12% of all experimental trials missed the target Since performance was monitored at all times by the physiotherapist, those trials that missed were repeated until correct and were not included for analysis.

In the 'NV conditions the subject was instructed to close his eyes on hearing the "Ready" warning signal over the intercom and to keep them closed over the trial duration. With the subject's eyes remaining shut the experimenter passively moved the limb back to the starting position and only once there was the subject permitted to open them.

In 'VR and 'VL conditions, a vertical screen was positioned in the saggital plane, perpendicular to the subject's shoulder girdle between the shoulder LED of the arm to be occluded and the head, such that when the head was central no movement of the screened arm could be seen by the subject. The screen did not interfere with arm or shoulder action. During practice trials the screen was used to allow both the subject and experimenter to gain familiarity with its deployment. In the course of the experiment it was positioned quickly and immediately prior to the first "Ready" signal.

Data Analysis

This followed the procedure of Experiment 4.1.

## Results

Since two of the differential viewing conditions were nested within the bilateral level of the factor Coordination, two ANOVA's were conducted on each subject's data: -

1) Arms x Vision x Co-ordination (2 X 2 x 2) to examine differences between unilateral and bilateral extension movements with and without the opportunity of continuous visual control(V, NV).

2) Arms x Vision (2 x 4) to examine bilateral actions when the amount of visual information is varied (V, NV, VL, VR).

### Simultaneity of Action

Means and standard deviations of initiation time (IT) are presented in Table 4.2.1. The presence of only one main effect of Arm in ANOVA (1) (MU: A, $F = 9.02$, $dF = 1,7$, $p<0.05$) reinforces the conclusion of Experiment 4.1 that the two arms start moving around the same time. On ANOVA (2) two subjects show main effects of arm (MY: A, $F = 21.40$, $dF = 1,7$, $p<0.01$ ;TO: A, $F=31.57$, $df=1,7$, $p<0.001$). Only one Arm x Co-ordination interaction was found (MY: AxC, $F = 6.70$, $dF = 1,7$. $p<0.05$) though once again this finding must be qualified by a lack of difference in IT between left and right unilateral movements.

Table 4.2.1
Mean (SD) Initiation Time (msecs)

| Movement Condition | Subjects MU | SM | MY | TO |
|---|---|---|---|---|
| LV | 242 | 535 | 373 | 376 |
| | (056) | (331) | (220) | (037) |
| LRV | 296 | 421 | 393 | 425 |
| | (046) | (194) | (136) | (074) |
| LRVL | 257 | 503 | 342 | 375 |
| | (038) | (244) | (930) | (101) |
| LRVR | 326 | 469 | 403 | 427 |
| | (107) | (142) | (1021) | (139) |
| LRNV | 368 | 632 | 372 | 433 |
| | (104) | (073) | (083) | (035) |
| LNV | 233 | 453 | 325 | 348 |
| | (099) | (172) | (117) | (039) |
| RV | 318 | 507 | 287 | 256 |
| | (101) | (155) | (085) | (034) |
| RLV | 341 | 611 | 505 | 345 |
| | (082) | (281) | (149) | (094) |
| RLVR | 317 | 462 | 496 | 365 |
| | (089) | (141) | (1092) | (120) |
| RLVL | 269 | 544 | 471 | 267 |
| | (073) | (312) | (998) | (027) |
| RLNV | 404 | 621 | 423 | 431 |
| | (138) | (259) | (125) | (032) |
| RNV | 340 | 607 | 347 | 357 |
| | (100) | (110) | (131) | (072) |

Table 4.2.1
Mean (SD) Initiation Time (msecs)

| Movement Condition | Subjects TU | MA |
|---|---|---|
| LV | 240 (037) | 181 (019) |
| LRV | 265 (074) | 183 (022) |
| LRVL | 256 (063) | 184 (018) |
| LRVR | 243 (056) | 174 (021) |
| LRNV | 230 (035) | 185 (021) |
| LNV | 282 (039) | 179 (019) |
| RV | 232 (034) | 172 (018) |
| RLV | 273 (094) | 178 (022) |
| RLVR | 269 (086) | 171 (026) |
| RLVL | 268 (041) | 177 (018) |
| RLNV | 262 (032) | 184 (018) |
| RNV | 253 (072) | 185 (023) |

Table 4.2.2
Mean (SD) Movement Time (secs)

| Movement Condition | Subjects MU | SM | MY | TO |
|---|---|---|---|---|
| LV | 0.998 | 1.667 | 1.208 | 1.295 |
| | (0.140) | (0.216) | (0.143) | (0.278) |
| LRV | 1.255 | 1.570 | 1.221 | 1.710 |
| | (0.111) | (0.384) | (0.134) | (0.268) |
| LRVL | 1.341 | 2.000 | 1.227 | 1.979 |
| | (0.155) | (0.378) | (0.271) | (0.285) |
| LRVR | 1.294 | 2.344 | 1.544 | 2.207 |
| | (0.292) | (0.572) | (0.309) | (0.418) |
| LRNV | 1.336 | 1.813 | 1.422 | 1.627 |
| | (0.501) | (0.678) | (0.235) | (0.262) |
| LNV | 0.968 | 1.950 | 1.216 | 1.305 |
| --- | (0.192) | (0.300) | (0.296) | (0.424) |
| RV | 1.315 | 3.021 | 2.058 | 1.855 |
| | (0.077) | (0.459) | (0.312) | (0.435) |
| RLV | 1.229 | 2.794 | 1.777 | 1.788 |
| | (0.139) | (0.445) | (0.163) | (0.397) |
| RLVR | 1.566 | 3.043 | 1.590 | 1.846 |
| | (0.354) | (0.599) | (0.300) | (0.386) |
| RLVL | 1.714 | 3.046 | 2.154 | 2.263 |
| | (0.442) | (0.575) | (0.223) | (0.533) |
| RLNV | 1.964 | 3.445 | 2.094 | 2.135 |
| | (0.384) | (0.615) | (0.439) | (0.454) |
| RNV | 1.837 | 2.851 | 2.163 | 1.858 |
| | (0.509) | (0.688) | (0.265) | (0.213) |

Table 4.2.2
Mean (SD) Movement Time (secs)

| Movement Condition | Subjects TU | MA |
|---|---|---|
| LV | 0.839 | 0.317 |
| | (0.107) | (0.022) |
| LRV | 1.009 | 0.337 |
| | (0.172) | (0.029) |
| LRVL | 1.093 | 0.311 |
| | (0.138) | (0.019) |
| LRVR | 1.091 | 0.329 |
| | (0.136) | (0.021) |
| LRNV | 1.049 | 0.250 |
| | (0.165) | (0.106) |
| LNV | 0.915 | 0.333 |
| | (0.077) | (0.033) |
| RV | 1.025 | 0.317 |
| | (0.178) | (0.018) |
| RLV | 0.916 | 0.308 |
| | (0.125) | (0.032) |
| RLVR | 1.091 | 0.321 |
| | (0.179) | (0.022) |
| RLVL | 1.318 | 0.322 |
| | (0.342) | (0.018) |
| RLNV | 1.087 | 0.291 |
| | (0.155) | (0.105) |
| RNV | 1.137 | 0.333 |
| | (0.180) | (0.029) |

Table 4.2.3
Correlations between the arms Sig: >.707

| SUBJECT | CONDITION | IT | TPV | FT |
|---|---|---|---|---|
| TU | RLV | +.397 | +.223 | +.807 |
| | RLNV | -.084 | +.489 | -.590 |
| | RLVR | -.152 | +.218 | -.046 |
| | RLVL | +.974 | +.513 | +.501 |
| SM | RLV | +.304 | +.391 | +.144 |
| | RLNV | +.368 | -.028 | -.655 |
| | RLVR | +.479 | +.432 | +.510 |
| | RLVL | +.664 | -.031 | +.279 |
| TO | RLV | -.043 | +.749 | +.878 |
| | RLNV | +.598 | -.321 | -.091 |
| | RLVR | +.471 | +.382 | +.518 |
| | RLVL | -.480 | +.493 | +.250 |
| MU | RLV | +.542 | +.373 | +.257 |
| | RLNV | +.620 | +.525 | +.113 |
| | RLVR | -.675 | +.757 | +.446 |
| | RLVL | -.625 | +.058 | +.466 |
| MY | RLV | -.066 | -.081 | -.043 |
| | RLNV | +.028 | +.490 | -.268 |
| | RLVR | +.261 | +.538 | +.262 |
| | RLVL | +.298 | +.308 | -.045 |
| MA | RLV | +.766 | +.983 | +.538 |
| | RLNV | +.327 | +.930 | +.662 |
| | RLVR | +.957 | +.164 | +.957 |
| | RLVL | +.792 | +.971 | +.853 |

Withdrawal of visual information concerning either both or one arm in the bilateral movements does not affect IT at all, as there are no main effects of Vision or interactions involving it in either ANOVA (1) or (2). In keeping with the results of the previous experiment the control subject MA shows no main effects or interactions on IT.

Table 4.2.2 contains the means of movement time (MT) and as with the IT ANOVA's, they reveal a similar picture to the previous experiment. There are main effects of Arm in both ANOVA's, except for subject GO in ANOVA (1) and subjects TU and TO in ANOVA (2). ANOVA (1): (MU: A, $F = 25.27$, $dF = 1,7$, $p<0.01$; SM: A, $F = 79.03$, $dF = 1,7$, $p<0.001$; MY: A, $F = 112.68$, $dF = 1,7$, $p<0.001$; TU: A, $F = 5.92$, $dF = 1,7$, $p<0.05$; TO: A, $F=30.54$, $df=1,7$, $p<0.001$). ANOVA (2): (MU: A, $F = 20.22$, $dF = 1,7$, $p<0.01$; SM: A, $F = 63.88$, $dF = 1,7$, $p<0.001$; MY: A, $F = 65.63$, $dF = 1,7$, $p<0.001$). Therefore, in general, movements of the affected arm even when performed at the same time as the unaffected arm, take longer to execute. As for IT there are no differences between the arms on MT for the normal subject MA. As in Experiment 4.1 correlations between arms on start time, time to peak velocity and finish time show consistent precise timing for the normal subject, but not the hemiparetic subjects (Table 4.2.3).

The lack of any main effect of Co-ordination or Vision x Co-ordination interactions in ANOVA (1) plus significant main effects of Vision in ANOVA (2) (MU: V, $F = 5.13$, $dF = 3.21$, $p<0.01$; SM: V, $F = 3.33$, $dF = 3.21$, $p< 0.05$; MY: V, $F = 10.09$, $dF = 3.21$, $p<0.001$; TU: V, $F = 4.54$, $dF = 3,21$, $p<0.05$; TO: V, $F = 6.92$, $dF = 3,21$, $p<0.01$) with no Arm x Vision interaction, demonstrates that impoverishment of visual information during the movement significantly increases the MT of both limbs in double movements, and when the arms are moving separately.

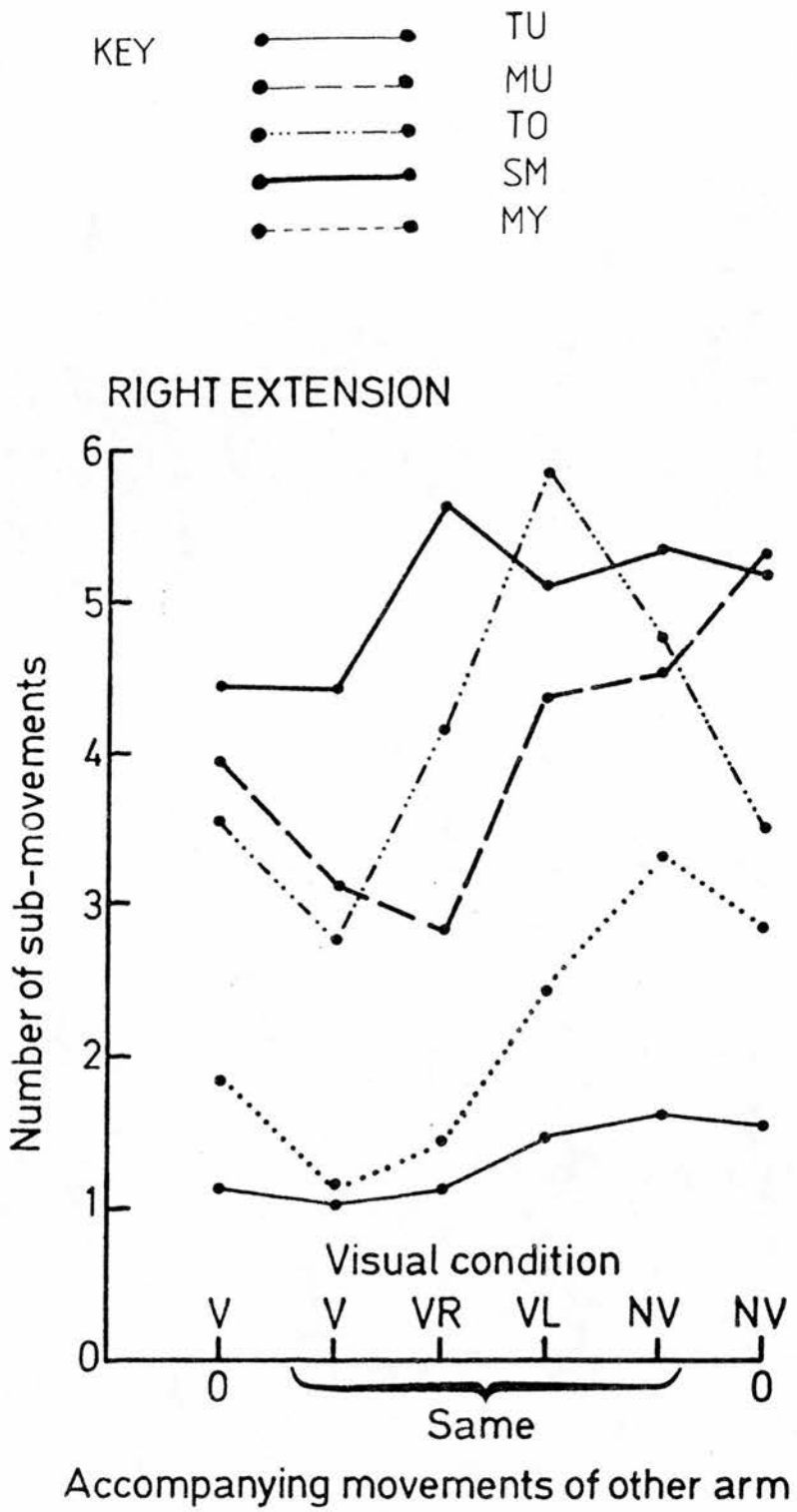
Trajectory

The mean number of submovements for each subject in each condition is plotted in Figure 4.2.1 and tabulated with standard deviations in Table 4.2.4. Since, as in the previous experiment, the normal subject consistently accomplished all the movements within one submovement, his data is not displayed.

Under full visual conditions, right (affected arm) extension in bilateral movements shows a drop in number of submovements for MY, TU, PU, and TO, with SM remaining the same compared to the unilateral condition (Figure 4.2.1). Concommitantly there is a slight non-significant rise in the number of submovements for left extension going from unilateral to bilateral, demonstrating as in

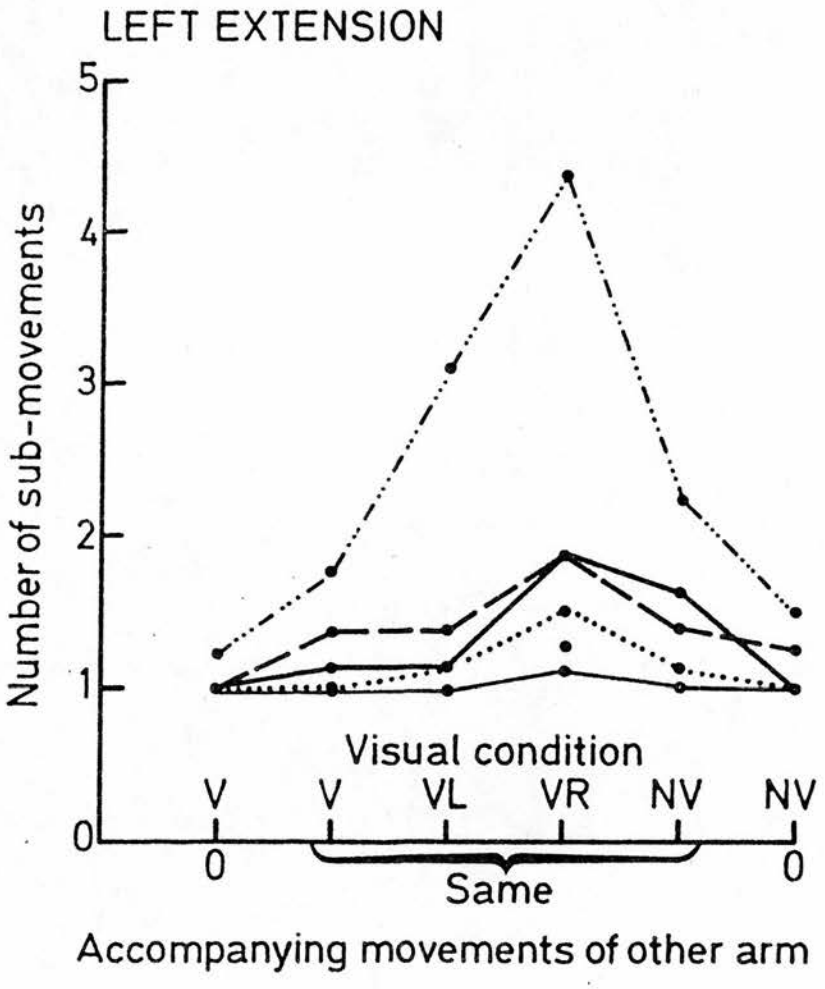
Table 4.2.4
Mean (SD) Number of Submovements

| Movement Condition | Subjects MU | SM | MY | TO | TU |
|---|---|---|---|---|---|
| LV | 1.00 | 1.00 | 1.00 | 1.25 | 1.00 |
| | (0.00) | (0.00) | (0.00) | (0.46) | (0.00) |
| LRV | 1.00 | 1.13 | 1.25 | 1.75 | 1.00 |
| | (0.00) | (0.35) | (0.46) | (0.88) | (0.00) |
| LRVL | 1.13 | 1.13 | 1.25 | 3.13 | 1.00 |
| | (0.35) | (0.35) | (0.46) | (1.13) | (0.00) |
| LRVR | 1.50 | 1.88 | 1.88 | 4.33 | 1.13 |
| | (0.76) | (0.99) | (0.99) | (1.30) | (0.35) |
| LRNV | 1.50 | 1.63 | 1.25 | 2.25 | 1.00 |
| | (0.35) | (0.76) | (0.46) | (0.71) | (0.00) |
| LNV | 1.75 | 1.13 | 1.25 | 1.50 | 1.00 |
| | (0.88) | (0.35) | (0.71) | (0.76) | (0.00) |
| RV | 1.75 | 4.38 | 3.88 | 3.50 | 1.13 |
| | (0.71) | (1.31) | (0.64) | (1.31) | (0.35) |
| RLV | 1.13 | 5.13 | 3.13 | 2.75 | 1.00 |
| | (0.35) | (0.74) | (0.35) | (0.71) | (0.00) |
| RLVR | 1.38 | 5.63 | 2.75 | 4.13 | 1.25 |
| | (0.52) | (1.41) | (0.89) | (1.46) | (0.46) |
| RLVL | 2.50 | 5.13 | 4.38 | 5.88 | 1.88 |
| | (0.93) | (0.64) | (1.06) | (2.10) | (0.84) |
| RLNV | 3.25 | 5.25 | 4.50 | 4.75 | 1.63 |
| | (0.71) | (1.55) | (1.20) | (1.17) | (0.52) |
| RNV | 2.75 | 4.38 | 5.25 | 3.50 | 1.50 |
| | (0.71) | (1.55) | (1.04) | (1.51) | (0.54) |




Same : Extension

Figure 4.2.1 Mean number of submovements.




Same : Extension

Figure 4.2.1 Mean number of submovements.

Experiment 4.1 that bilateral interaction between the limbs tends towards a consensus under full visual conditions. Also, following that experiment, this is generally reflected in within arm comparisons, since all subjects show a significant main effect of Arm on both ANOVA's. ANOVA (1) ;(MU: A, F = 59.97, dF = 1,7, $p<0.001$; SM: A, F = 276.34, dF = 1,7, $p<0.001$; MY: A, F = 288.00, dF = 1,7, $<0.001$; TU: A, F = 15.91, dF = 1,7, $p<0.01$; TO: A, F = 58.50, dF = 1,7, $p< 0.001$) and ANOVA (2); (MU: A, F = 11.67, dF = 1,7, $p<0.05$; SM: A, F = 250.19 dF = 1,7, $p<0.001$; MY: A, F = 119.95, dF = 1,7, $p<0.001$; TU: A, F = 18.78, dF = 1,7, $p<0.01$; TO: A, F = 42.56, dF = 1,7, $p< 0.001$).

Only MU showed no significant difference ($p>0.05$) comparing the two arms on these two conditions (Scheffe test) and MU, MY and TO show significant differences between bilateral right extension and unilateral right extension. However all subjects, except TO on ANOVA(1) and SM on ANOVA (2), show main effects of vision on ANOVA (1): (MU: V, F = 33.64, dF = 1,7, $p< 0.001$; SM V, F = 5.90, dF = 1,7, $p<0.05$; MY: V, F = 12.60, dF = 1,7, $p<0.01$; TU: V, F = 28.00, dF = 1,7, $p<0.01$) and ANOVA (2): (MU: V, F = 18.55, dF = 3.21, $<0.001$; MY: V, F = 10.88, dF = 3,21, $p<0.001$; TO: V, F = 21.83, dF = 3,21, $p< 0.001$).

Individual comparisons reveal that right extension

without vision results in a significant increase in submovements in all subjects, except TO, that is not found in comparisons for the left arm. Also, as can be seen from the Figure, the reduction in submovements composing bilateral affected arm extension is lost when vision is completely withdrawn. This is reflected, specifically for the right arm by Arm x Vision interactions in three of the five subjects on ANOVA (1): (MU: A x V, $F = 19.42$, $dF = 1,7$, $p<0.01$; MY: A x V, $F = 7.61$, $dF = 1,7$, $p<0.05$; TU: A x V, $F = 28.00$, $dF = 1,7$, $p<0.01$).

Notably perhaps, SM and TO do not exhibit this interaction due to a rise in number of submovements in the performance of the unaffected partner in bilateral movements when vision is withdrawn. Following from this point, the presence of only one Arm x Vision interaction in ANOVA (2) (MU: A x V, $F = 7.83$, $dF = 3.21$, $p<0.01$) indicates the potentially deleterious effects on unaffected limb performance of coupling the arms when conditions do not permit adequate ongoing control of action through visual monitoring. However, on the points covered in this paragraph, only TO shows significant differences on left extension, among the four bilateral conditions, as evidenced by a lack of main effect of Arm in ANOVA(2) and a main effect of Co-ordination in ANOVA(1): (TO: C, $F=11.97$, $df=1,7$, $p<0,05$).

When in the 'VR condition subjects can see their affected, but not unaffected, limb throughout the movement, the consensus effect is significantly evident in two out of the five subjects (MU and MY) as opposed to being statistically present in three when vision of both arms is permitted. But in the 'VL condition it is lost completely. Therefore, although facilitation of affected arm performance can in some way be achieved by having only somatic proprioceptive information concerning the position of the unaffected arm, visual monitoring of the affected arm is vital throughout the movement.

Subjects MY and MU show significant main effects of Co-ordination in ANOVA (1) for IT, (MU: C, $F = 20.44$, $dF = 1,7$, $p<0.01$; MY : C, $F = 6.49$, $dF = 1,7$, $p<0.05$) and demonstrate the pattern of rise in IT, with reduction in number of submovements, that would be expected if the facilitation of affected arm extension in bilateral movements was due to interaction during the planning phase of the movement. This, however, has to be balanced against the lack of main effects of Vision and interactions of Vision and Arm, so prevalent in the submovement data. There is a parallel between MT and the number of submovements on these points, with MT shortening as the number of submovements reduces. Also, the necessity of continuous visual monitoring of the affected arm during performance and the trend for the number of submovements in unaffected limb performance to

increase in double movements when complete visual information is not available, as shown significantly by T0, tends rather to favour a hypothesis of the consensus effect occurring whilst the limbs are actually moving.

## Discussion

The data of this experiment replicate the findings of Experiment 4.1 for bilateral interaction and affected arm extension: this movement is facilitated by simultaneous homologous activity of the healthy limb. However, this facilitation can only occur when full visual monitoring of affected arm performance is permitted over the course of the action. This lends support to a conception of the effect as being due to active communication between ipsilateral and contralateral control processes during movement production. That it is the product of an interaction between voluntary control processes, emerges clearly from the double limb conditions where vision was manipulated or removed. If bilateral interaction was simply of a reflex nature, then selective or complete withdrawal of vision should not affect control.

The worsening of performance found for both arms, shown so powerfully by subject TO, is probably indicative of the conditions prevailing during the Cohn (1951), Hausmanowa-Petrusewicz (1959) studies, i.e that their recorded movements occurred in a non-functional context with little provision for continuous control. As both this and the prior experiment have demonstrated, when conditions are adequate for control, bilateral interaction can result in facilitation of affected arm

movement, without appreciable detriment in healthy limb performance.

## Experiment 4.3 - Reaching to a moving target

### Introduction

Following from the results of Chapter III, Experiment 4.2 indicated that what may be happening in the bilateral case is that through a combination of visual comparison of the two actions and the additional ipsilateral efference to the involved arm, the timing of muscle activity is improved in the affected arm.

In the case of a stationary target, where there are no external temporal constraints, timing is intrinsic to the system. This led to the hypothesis that by imposing an extrinsic timing demand upon hemiparetic movement bilateral synchrony might be enhanced with a greater bilateral effect upon performance. This experiment was designed to examine this question.

## Method

### Design

As in Experiment 4.2, all movements were extension movements, however in this experiment the subject had to reach to a ball instead of striking a target with a dowel. Movement conditions were unilateral/bilateral with two target conditions (stationary/moving) resulting in six combinations of conditions:-

Unilateral Conditions

1) RS - extension of the right arm to a stationary ball.

2) LS - as (1) for left arm.

3) RM - extension of the right arm to a moving ball.

4) LM - as (3) for left arm.

Bilateral Conditions

5) RLS - extension of both arms to a stationary ball.

6) RLM - extension of both arms to a moving ball.

### Subjects

Four of the subjects who took part in the previous experiment participated here : MU, SM, MY and TO. Normative data for this experiment was again provided by MA.

### Apparatus

A white plastic soccer ball (20cm in diameter) constituted the target. In stationary conditions it rested 24 cms from the subject's edge of the table. Two LED'S fixed laterally to the equator of the ball and

equidistant from the subject served as indicators that the hand was on target. In order that it did not roll on impact, the ball was cradled in a circular tin 3cms in depth and 8cms in diameter, which was fixed to the table.

In moving target conditions the ball was rolled down a track, in the subject's tranverse plane, towards his face, (Figure 4.3.1). The track was 1.72m long and consisted of two aluminium rods 1.5cms in diameter and mounted 7.5cms apart. The track ended level with the subject's edge of the table and began 9 cms above that height, resulting in a end to end ball movement time of up to approximately 2.5 secs. A strip of surgical tape spanned the underside of the rods 24cms up the track from the subject. A further 10cms towards the top of the track was another strip. Fixed to the middle of each of these strips was an LED, which acted to chart the latter portion of the ball's progress. The strip nearer to the subject also functioned as a target line for striking or trapping the ball.

As in the previous experiments, a non-continuous LED was mounted in constant view of the camera and linked to the pulse generator circuit. Since the dowels were not used, thin brass foil connected to the circuit was moulded and fixed around the ulnar edge of the hand. At the start of a trial the foil broke contact

Figure 4.3.1 - The track and ball.

with the home bases and registered the start of the movement.

Procedure - Positioning of the subject and placement of LED'S on the limbs was as previously described. In stationary conditions the subject was simply asked to strike the ball with his hand or hands as quickly as possible. The only other stipulation was that, on impact, his hands should cover the laterally mounted LED'S. After each of these trials the LED'S were re-positioned if necessary.

The instructions for moving ball conditions differed slightly depending on whether the condition was single or double limb. In unilateral trials the subject was requested to knock the ball off the track - this required a minimum of force. When the two arms were used, the goal was to trap the ball between the hands. In both cases it was emphasised that an attempt should be made to contact the ball as it was crossing the strip of surgical tape nearer to the subject. In addition to checking the subject's posture, positioning, and performance in these trials, the physiotherapist acted as a 'goalkeeper' intervening to prevent the ball hitting the subject's face if he fumbled or missed it. "Missed" trials were repeated.

Data

The same data was extracted as in the other experiments. Estimation of MT was aided by occlusion of the LED'S

placed on the stationary ball and the track. In moving ball trials visual inspection of the arm trajectory and its subsequent velocity profile revealed that when the hand made contact with the ball, the wrist jolted sharply and momentarily backwards. Using this observation completion of movement was defined as being at the end of the submovement prior to this jolt.

## Results

### Simultaneity of Action

The pattern of simultaneity in bilateral movements differs from the previous two experiments. Table 4.3.1 lists means and standard deviations of initiation time (IT) for each subject across all the conditions. From 3-way (Arm x Ball x Co-ordination) repeated measures ANOVA's carried out on each subject's data, no main effect of Arm is found. Thus replicating the finding that, in general, the arms start moving at around the same time.

However, when movement time (MT) data (Table 4.3.2) is analysed, although each subject demonstrates a main effect of Arm, two subjects show significant Arm x Co-ordination interactions (SM: A x C, $F = 7.33$, $dF = 1,7$, $p<0.05$; MY: A x C, $F = 10.74$, $dF = 1,7$, $p<0.05$;), and a third subject approaches significance on this interaction (MU: A x C, $F = 4.83$, $dF = 1,7$, $p = 0.064$). Individual comparisons between means (Scheffe tests) show this to be due to a lowering of MT in the affected arm when it is moving with the unaffected arm. Therefore, although overall the movement time for the affected limb is greater than the unaffected, it is significantly reduced in the bilateral condition.

Table 4.3.1
Mean (SD) of Initiation Time

| MOVEMENT CONDITION | SUBJECTS MU | SM | MY | TO | MA |
|---|---|---|---|---|---|
| LS | 242 | 372 | 386 | 305 | 192 |
| | (031) | (128) | (110) | (089) | (016) |
| LRS | 226 | 372 | 264 | 453 | 179 |
| | (041) | (158) | (031) | (169) | (015) |
| LM | 399 | 490 | 573 | 1108 | 1648 |
| | (327) | (188) | (183) | (288) | (115) |
| RLM | 384 | 489 | 528 | 1120 | 1682 |
| | (168) | (170) | (280) | (453) | (100) |
| RS | 278 | 229 | 256 | 351 | 178 |
| | (123) | (063) | (066) | (100) | (020) |
| RLS | 234 | 570 | 238 | 418 | 178 |
| | (030) | (265) | (021) | (139) | (021) |
| RM | 382 | 426 | 439 | 1055 | 1631 |
| | (218) | (251) | (234) | (256) | (086) |
| RLM | 347 | 559 | 669 | 1229 | 1657 |
| | (111) | (160) | (265) | (179) | (081) |

Table 4.3.2
Mean (SD) of Movement Time

| MOVEMENT CONDITION | SUBJECTS MU | SM | MY |
|---|---|---|---|
| LS | 0.823 | 1.292 | 1.126 |
| | (0.115) | (0.291) | (0.254) |
| LRS | 0.905 | 1.335 | 1.145 |
| | (0.162) | (0.208) | (0.137) |
| LM | 1.314 | 1.292 | 1.164 |
| | (0.310) | (0.266) | (0.136) |
| LRM | 1.260 | 1.258 | 1.273 |
| | (0.205) | (0.234) | (0.074) |
| RS | 1.309 | 2.403 | 2.031 |
| | (0.205) | (0.259) | (0.322) |
| RLS | 1.706 | 1.863 | 2.042 |
| | (0.316) | (0.493) | (0.140) |
| RM | 1.392 | 1.393 | 1.569 |
| | (0.260) | (0.268) | (0.371) |
| RLM | 1.494 | 1.339 | 1.205 |
| | (0.289) | (0.114) | (0.069) |

Table 4.3.2
Mean (SD) of Movement Time

| MOVEMENT CONDITION | SUBJECTS TO | MA |
|---|---|---|
| LS | 1.050 | 0.473 |
| | (0.154) | (0.042) |
| LRS | 1.170 | 0.521 |
| | (0.265) | (0.042) |
| LM | 1.071 | 0.733 |
| | (0.358) | (0.050) |
| LRM | 1.101 | 0.798 |
| | (0.456) | (0.074) |
| RS | 1.643 | 0.461 |
| | (0.276) | (0.051) |
| RLS | 1.354 | 0.510 |
| | (0.157) | (0.033) |
| RM | 1.176 | 0.701 |
| | (0.253) | (0.096) |
| RLM | 0.970 | 0.806 |
| | (0.171) | (0.083) |

Table 4.3.3
Correlations between the arms Sig: >.707

| SUBJECT | CONDITION | IT | TPV | FT |
|---|---|---|---|---|
| MU | RLS | +.925 | +.800 | +.598 |
| | RLM | +.234 | +.309 | +.395 |
| SM | RLS | -.374 | -.488 | +.173 |
| | RLM | +.422 | +.326 | +.642 |
| MY | RLS | -.398 | +.546 | +.034 |
| | RLM | +.262 | +.524 | -.137 |
| TO | RLS | -.061 | +.367 | +.014 |
| | RLM | +.356 | +.805 | +.638 |
| MA | RLS | +.619 | +.840 | +.533 |
| | RLM | +.378 | +.941 | +.856 |

As in the previous experiments none of the hemiparetic subjects show precise synchronisation of bilateral activity as measured by correlation of IT, time to peak velocity, and finish time (Table 4.3.3). The normal subject MA does, and also shows no significant effects on IT or MT for differences between arms in double arm movements. However as with the normal subject (AL) in the first experiment, MA shows a significant increase in MT when going from unilateral to bilateral: (MA: C, $F=36.58$, $df=1,7$, $p<0.001$).

Trajectories

Since the normal subject MA consistently accomplished all conditions within one submovement, only submovement data for hemiparetic subjects is depicted in Figure 4.3.2, with means and standard deviations tabulated in Table 4.3.4. As before, a main effect of Arm for all subjects ((SM: A, $F = 221.87$, $dF = 1,7$, $p<0.001$; MY: A, $F = 196.87$, $dF = 1,7$, $p<0.001$; MU: A, $F = 105.00$, $dF = 1,7$, $p<0.001$; TO: A, $F=94.95$, $df= 1,7$, $p<0.001$ ) indicates a general tendency for the affected limb to produce more submovements than the unaffected arm.

Subjects SM and MU took part in all three experiments reported in this Chapter, but their performance on ball interception differs from that in the other two experiments. MU shows a complete reversal of the effect of bilateral interaction. She is the only subject, in any of these experiments, to show a

Table 4.3.4
Mean (SD) of Number of Submovements

| MOVEMENT CONDITION | SUBJECTS MU | SM | MY | TO |
|---|---|---|---|---|
| LS | 1.00 | 1.88 | 1.38 | 1.13 |
| | (0.00) | (1.13) | (0.52) | (0.35) |
| LRS | 1.00 | 2.50 | 1.13 | 1.50 |
| | (0.00) | (0.53) | (0.35) | (0.53) |
| LM | 1.38 | 1.50 | 1.13 | 1.38 |
| | (0.52) | (0.76) | (0.35) | (0.52) |
| RLM | 1.38 | 1.13 | 1.13 | 1.13 |
| | (0.74) | (0.35) | (0.35) | (0.35) |
| RS | 1.63 | 5.00 | 4.13 | 3.89 |
| | (0.52) | (0.93) | (1.25) | (1.73) |
| RLS | 2.75 | 3.50 | 4.13 | 2.36 |
| | (0.89) | (1.20) | (0.99) | (0.92) |
| RM | 1.88 | 2.88 | 2.38 | 3.00 |
| | (0.64) | (1.13) | (0.92) | (0.93) |
| RLM | 2.25 | 2.38 | 1.63 | 1.63 |
| | (0.71) | (1.06) | (0.74) | (0.74) |

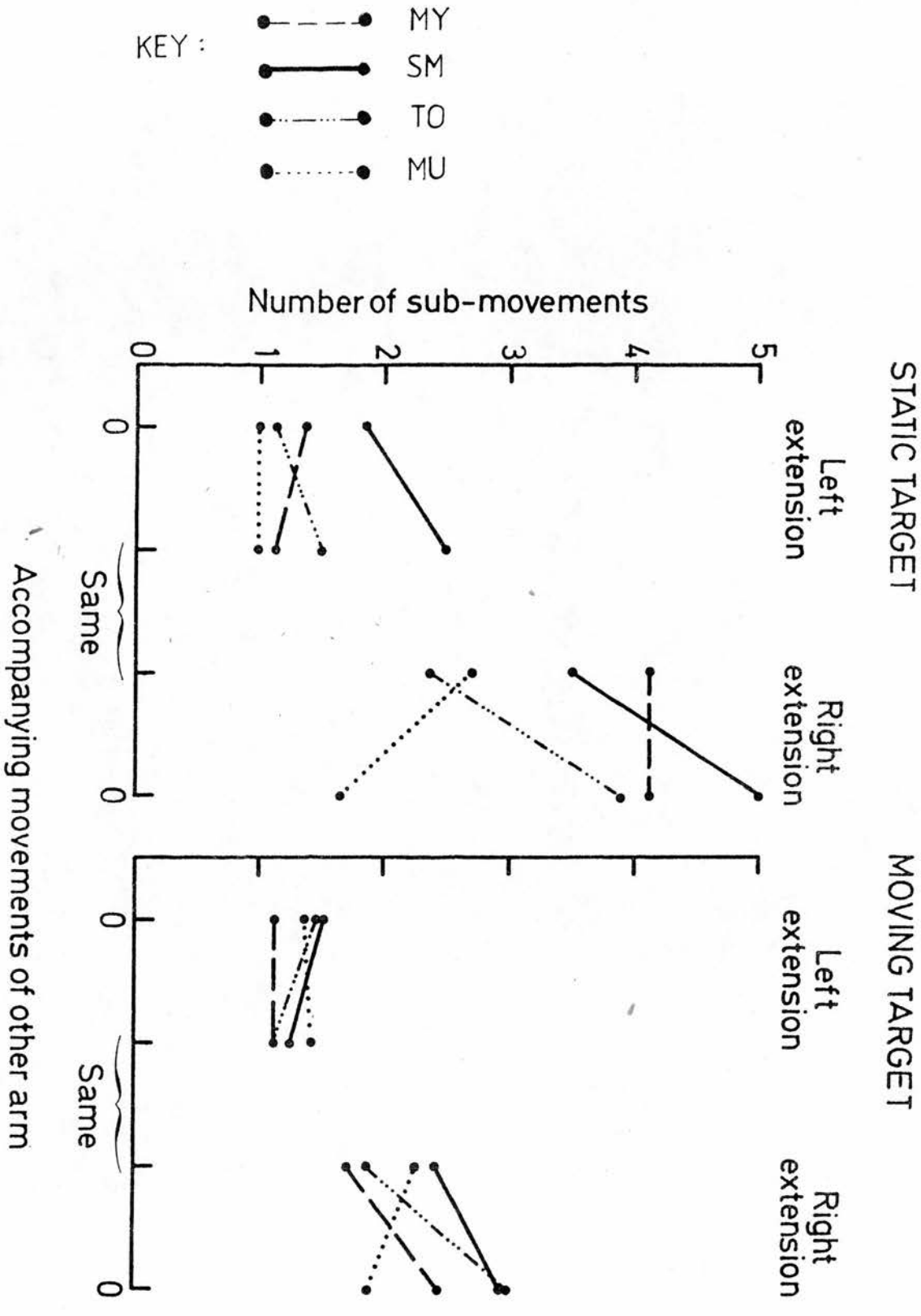



Figure 4.3.2 Mean number of submovements.

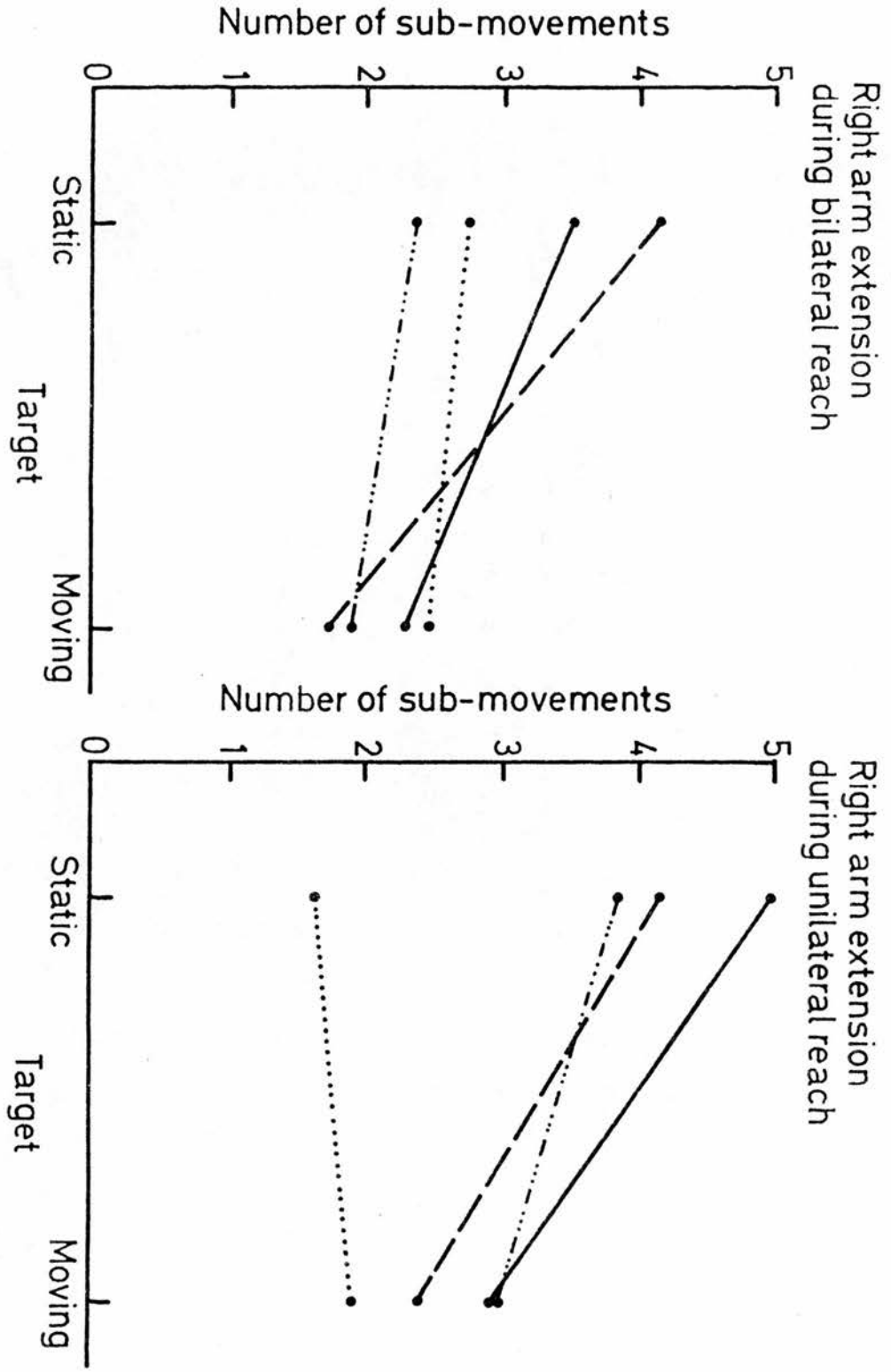



Figure 4.3.3 Comparing the effect of a static or moving target on mean number of submovements.

significant increase in number of submovements going from unilateral to bilateral interception (MU: C, $F = 5.73$, $dF = 1,7$, $p<0.05$) with the affected arm (MU: A x C, $F = 12.60$, $dF = 1,7$, $p<0.01$). In the light of the results with the dowel placing task this is quite puzzling. However, the ball catching task demands a greater degree of shoulder elevation, and the subject was weak in this movement. When she first took part in this study, MU was two years and three months post-CVA. Her records indicated that initially she had very poor and slow recovery dominated by moderate spasticity. Although the Table 4.1.1 shows shoulder activity at 3+, a fuller assessment revealed poor shoulder girdle protraction/retraction with protraction as a particular problem. She had, therefore, a limited range of scapula movement. When moving one arm she could have used trunk action to aid limb elevation, but when moving both arms this would have been more difficult given the task demands. MU was the only subject found to have this problem with scapula movement and it was more than likely a consequence of her long term spasticity.

Subject SM, although not exhibiting a deterioration of right arm performance in bimanual conditions, shows a clear effect for the first time in ball interception(SM: A x C, $F = 7.99$, $dF = 1,7$, $p<0.05$). Individual comparisons on this facilitation show it to be due to the presence of significant change in right extension only.

Subject TO also follows this pattern with (TO: A x C, F = 7.41, dF = x1,7, $p<0.05$) qualifying a main effect of C (TO: C, F=1,7, $p<0.05$). Subject MY follows this trend for the moving ball while showing no detrimental effects of bimanual responses upon right arm performance in the static condition, but unlike TO, MY showed no significant effect of bimanual coordination as such.

Discounting MU's results, it may be generally concluded that bilateral activity has again facilitated affected arm performance. However, in this experiment the 'consensus effect' was not evident only SM in the static condition shows a non-significant trend for a rise in number of submovements in extension of the left (unaffected) arm. The link between a reduction in MT and reduction in number of submovements established in the prior experiment is upheld.

Static versus Moving Target

As Figure 4.3.2 shows, there is a general reduction in number of submovements across the static to moving targets for every subject except MU on unilateral right extension. Breakdown of main effects of ball in SM, TO and MY (SM: B, F = 43.75, dF = 1,7, $p<0.001$; MY: B, F = 40.50, dF = 1,7, $p<0.001$; TO: B, F = 14.91, dF = 1,7, $p<0.01$) and an Arm x Ball interaction for MY ( MY: A x B, F=89.60, df=1,7, $p<0.001$) show that across the factor Co-ordination there are significantly less submovements in right extension when the subject is reaching to a

moving target.

The presence of Arm x Ball interactions in MT data of Table 4.3.2 for all hemiparetic subjects (MU: A x B, $F = 16.68$, $dF = 1,7$, $p<0.01$; SM: A x B, $F = 21.06$, $dF = 1,7$, $p<0.01$; TO: A x B, $F = 14.27$, $dF = 1,7$, $p< 0.01$; MY: A x B, $F = 58.72$, $dF = 1,7$, $p<0.001$) reinforces the finding for Co-ordination effects in this and the previous experiment, of a reduction in movement time with a reduction in number of submovements. A static/moving target effect on MT is shown in the normal subject in the reverse direction (MA: B, $F=191.44$, $df= 1,7$, $p<0.001$).

The normal subject MA shows a main effect of Ball (MA: B, $F = 4580$, $dF = 1,7$, $p<0.0001$) with a greater initiation time for movements to the intercept the moving ball. As might have been expected he was delaying execution of action in the attempt to precisely trap the rolling ball as it crossed the target line with as fast a movement as possible. The presence of a main effect of Ball in all hemiparetic subjects (MU: B, $F = 15.45$, $dF = 1,7$, $p<0.01$; TO: B, $F = 180$, $dF = 1,7$, $p<0.001$; MY: B, $F = 43.24$, $dF = 1,7$, $p<0.001$; SM: B, $F = 5.88$, $dF = 1,7$, $p<0.05$) demonstrates quite clearly that they also were attempting to do this.

## Discussion

In the ball interception task the goal for arm movement was made more precise by introducing temporal constraints. This did not lead to tighter synchronisation between the affected and normal arms, but it did produce a smoother hemiparetic trajectory in both the bilateral and unilateral reaching. It may be concluded that this supports arguments concerning the importance of a functional context for hemiparetic actions, both when the paretic arm is moving on its own and when it is coordinated with the unaffected arm. As task demands are made stricter and the goal of movement becomes more precise, increased precision of control is required. However, at the same time the visual information available to the subject specifies more clearly how his actions should be timed. Prior research with normal subjects in a situation analagous to the moving ball task, demonstrated that the whole movement was not pre-programmed (Lee et al, 1983). Rather timing of interception was continuously guided by optical information specifying the time to contact of limb with target. This information was available to the patients in this experiment. If their faulty movement is due to poor timing of muscle activity, then providing them with the necessary extrinsic information for timing should aid performance. The results indicate that this was indeed

the case.

The increased initiation time found for the hemiparetic subjects in the moving ball conditions also helps explain the improved level of performance. In all three of the experiments in this Chapter the instructions stress speed and accuracy of movement. In the first two experiments and the static ball condition of the third, subjects interpreted this to mean quickness in both leaving the home-base and executing the movement. In the moving ball condition, to be fast and accurate necessitated delaying the start of action. This resulted in more time being available for the planning of action and could have allowed more facilitation through bilateral communication to occur during the preparatory phase. However there is a contrary effect of the moving ball upon the time to make the movement between the normal subject MA and those hemiparetic subjects (SM, MY and TO) who show facilitation of performance with the affected arm. In these three subjects MT significantly decreases whilst for MA it increases.

Given MA's ability to prepare action through normal tuning, the stationary target would facilitate the task demand of speed by reducing time uncertainty, thus permitting a more efficiently pre-programmed movement to the target. For MA the time to initiate movement becomes more variable the moving target (Static range: 16 - 21

msecs; Moving range: 81 - 115 msecs) reflecting an increased uncertainty in programming (Schmidt, 1969). Since hemiparetics appear to be defective in the advance programming of actions, and Experiments 4.1 and 4.2 revealed little effect on IT, it seems more likely that the moving ball facilitated performance by assissting a continuous mode of feed-back control, rather than by improving preparation.

## General Discussion

In the introduction to this Chapter two differing predictions were advanced concerning the effects upon motor performance of simultaneous activity in the normal and hemiparetic limb. The 'compromise hypothesis' predicted that no improvement in hemiparetic action would ensue and that the level of normal limb performance would deteriorate to match it. The 'consensus hypothesis' proposed that interaction between the control processes governing the activity of each limb would result in performance tending towards an intermediate level for both limbs.

Although two prior studies (Cohn, 1951; Hausmanowa-Petrusewicz, 1959) strongly favoured the compromise hypothesis, with the latter study additionally predicting a worsening of hemiparetic limb performance, the evidence presented here is more in keeping with the consensus hypothesis. Moreover, the effect upon normal limb activity was for most subjects less than expected from that hypothesis. However, perhaps more important is the finding that over the three experiments, simultaneous, homologous, bilateral activity tends to ameliorate affected arm performance in those patients whose unilateral performance levels differ ed significantly.

In the discussion following Experiment 4.1 a model of bilateral interaction was presented based on the work of Preilowski (1975) and Marteniuk and McKenzie (1980) to account for the contrary effects of simultaneous homologous versus non-homologous contralateral movements upon affected arm extension. Since the experiments in Chapter III demonstrated that stroke patients experience particular difficulty in executing extension movements about the elbow joint because their flexors tend to cocontraction, the presence of an additional ipsilateral impulse to extension could help prevent co-contraction at the start of the movement and in reducing its influence during overt activity. But when the ipsilateral control processes specify flexion, then co-contraction would increase resulting in more inhibition of extension.

The conception that these principles of organisation are essentially those underlying normal voluntary motor activity is supported by the evidence on the active nature of the interaction between the limbs. Cohn (1951) demonstrated this when he found an influence of active but not passive movement of the affected arm on healthy limb performance. The findings with the rolling ball showed that varying the parameters known to be involved in normal motor control produces differential effects on interaction between the limbs. The phenomenon discussed appears to be a higher level interaction between control processes (Cohn, 1951; Hausmanowa-Petrusewicz, 1951) and

not the synkinesia observed following hemispherectomy or from lateralised lesions in general (Cernacek, 1961; Zulch and Muller, 1967). The absence of synchronisation between movement elements in the limbs in the stroke patients who took part in this study would rule out a simple synkinetic explanation.

The descending pathways to the spinal cord are compromise both ipsilateral and contralateral brain influences on the musculature of one body half. Cortical output passing through the intact internal capsule can act upon the spinal processes governing the affected side, either directly, via the ipsilateral routes, or indirectly, through interaction between contralateral tracts at the subcortical level (Brodal, 1962; Kuypers, 1973). This results,at the spinal level, in the potential for normal supra-spinal activity to counteract or modulate the abnormal effects of the lesion transmitted by contralateral projection. A broad conclusion from studies of the functional organisation of the ipsilateral cortico-spinal pathways, is that these direct and indirect means of influence affect proximal arm action and complex arm-hand movements, but do not control individual hand or finger movements. The pattern of recovery in arm function documented by Twitchell (1951), occurs in a proximal to distal manner with frequent loss of return of appreciable hand or individual finger control. Also, the movements pass through a

holokinetic phase (gross movement control) before becoming more idiokinetic (fine movement control). It would appear that the processes proposed as underlying the results of the present experiments are the same as those that could constitute the neural mechanisms of recovery.

The experiments in this chapter underscore the importance of visual monitoring of affected arm performance. Experiment 4.2 indicated that for bilateral facilitation to occur sight of the affected arm is necessary. Even in the presence of a potential bias towards extension, visual tuning is necessary for smoother, faster movement. In addition the results of Experiment 4.3 demonstrate that by adding spatio-temporal constraints upon hemiparetic movement, performance can be improved. The key to this effect lies in the additional visual information available to the subject during movement execution.

# CHAPTER V

## Summary and Conclusions

The experiments reported in this thesis were guided by three questions:-

1) Is visual guidance during movement of benefit to the stroke patient and, if so, how does vision function to improve control?

2) What is the state of communication between the two sides of the body after a stroke that causes one half of the body to suffer partial or complete paralysis?

3) Is spasticity a problem in hemiparetic motor control?

In answer to question (1), a general conclusion is that visual guidance is of benefit to control of the hemiparetic arm. Sight of performance results in more accurate, smoother, and faster movement. The experiments in Chapter II indicated that vision serves a useful propriospecific (Experiments 2.1 and 2.2) and expropriospecific (Experiments 2.2 and 2.3) function in the control of extension of the affected arm. The similar pattern of directional error or bias which emerged from the vector analysis in these experiments suggests that these two functions are intimately linked. Vision affords a realistic appreciation of the position of the arm to both the rest of the body and externally defined targets. It therefore appears that stroke produces a miscalibration of somatic proprioception and that during recovery vision acts to recalibrate the

system.

From Chapter III two levels of functon of visual control can be tentatively proposed:-

1) Macrospecific

2) Microspecific

The macrospecific function of vision operates to ensure that movement is sustained in order to achieve the intended goal. It can be defined as operating at a "gross" level of control which ensures the limb achieves the movement target. The operation of this function explains the results in Chapters II and III which demonstrated undershooting of the target position when the limb was not visible. Precisely what may cause undershoot remains an open question. Two hypotheses were advanced to explain how, in the absence of vision, the patient may be misinformed as to the motoric results of his efforts:-

a) Through over-active antagonist Ia afferents.

b) Through a mismatch between expected and necessary efferent drive.

Although most of the evidence presented favours (a), there is still the possibility that (b) creates a problem in control as well. Following Experiment 2.2, further work is planned to attempt to resolve this issue.

It seems therefore that the macrospecific function of vision is concerned with matching an intended target

position with the end-point of movement. At this level of control continuous visual guidance may not be necessary. Experiment 2.3, which examined the role of knowledge of results (KR) in movement control, concluded that continuous visual guidance is necessary for accurate target attainment. However it must be noted that this conclusion was based on a group mean which may have been biased by the inability of two right hemisphere lesioned patients to utilise KR. In Experiment 4.2 all the patients studied had left hemisphere lesions and although the effective target size was larger than in Experiment 2.3, they were all able to be trained to hit the target using KR. Further study is therefore necessary to determine whether at the macrospecific level of control continuous visual guidance is necessary, particularly for patients with left hemishere lesions.

At the microspecific level, it can be argued that visual monitoring throughout the act is essential. Distinct from the macrospecific level, the microspecific level of vision is postulated to account for the effects vision has on the kinematics of movement. The increased mean submovement distance and velocity of extension, found in the visual condition of Experiment 3.1, implies that the visual system is registering velocity or acceleration of the arm during movement and that this information is used to improve control. The EMG data in that experiment, and, in Experiment 3.3, the improved

ability of the arm to switch from flexion to extension when the limb can be seen, suggest that visual information contributes to the supraspinal modulation of the timing of muscle activity. This conclusion is supported by the results of Experiment 4.3 where the provision of extrinsic timing information elevated the quality of movement.

Following Yanagisawa et al (1976) and Miller and Hammond (1982) in the concluding discussion of Chapter III, it was speculated that during extension of the affected elbow visual information is used by the patient in an attempt to try and reorganise an imbalance, at the interneuronal level, in reciprocal interaction between the agonist and antagonist. It must be emphasised that this conclusion is largely speculative and awaits more sophisticated neurophysiological examination.

The above discussion offers an answer to question (3). If an imbalance in reciprocal control of agonist/antagonist activity can be identified as the key cause of an imbalance in tone, then the consistently observed inferior performance of extension compared with flexion, suggests that spasticity is a problem in hemiparetic motor control. However the results of Chapter III indicate that it is only potentially a problem in the control of extension and not flexion. Two points must be made regarding this issue:-

1) Since spasticity was not objectively measured in any of the experiments reported in this thesis, its effects on movement can only be inferred from the difficulty the patients had in extending rather than flexing the affected arm.

2) Following Brunnstrom (1970), it could be argued that since all the patients studied could extend the elbow and achieve target criteria with visual monitoring, they were at a relatively advanced stage of recovery. Therefore the higher level of control found in flexion movements in Chapter III could indicate that the patients had already progressed from a stage where spasticity is a problem for flexion.

Point (2) leads to a general comment regarding any conclusion from this thesis. Any statement on hemiparetic motor control may only be applicable to the level of recovery that was required before the subject could perform the experimental tasks. Currently the author is engaged in a study with colleagues at Cambridge to investigate how the issues raised in this thesis apply during the course of the recovery process.

Regarding question (2), the results of Chapter II indicated that the processes controlling the two halves of the body are in communication with each other. However, in the absence of vision, internal communication alone appears inadequate to overcome motor defects in the paretic limb (Experiments 2.2 and 4.2). Evidence

presented in Chapters III and IV demonstrated both detrimental (Experiment 3.4) and beneficial (Experiments 4.1, 4.2 and 4.3) effects on paretic arm performance may arise from bilateral interaction between the processes controlling the two arms. From Experiment 4.2 it should be noted that any interaction of benefit to the affected arm is dependent on visually based knowledge regarding where the limb is and how it is moving.

In summary, the principal problems facing the patient appear to be:-

1) He seems unable to adequately preprogram movement of the affected limb and therefore has to tune the action as it is progressing.

2) Without visual monitoring, he is hindered in this task by unreliable somatic information regarding the position of the arm and how it is moving.

3) He also has a problem with timing of muscle activity.

Bearing in mind the warning that the results presented may be limited to a particular stage of recovery, there are several implications which emerge from this thesis, for the treatment of hemiparesis:-

1) Visual monitoring of his actions by the patient is of paramount importance and should be encouraged.

2) The vector analyses in Chapter II suggest that tests of proprioceptive dysfunction should contain a direction component.

3) Visual information is useful in an expropriospecific

as well as propriospecific sense. Given the difference in performance observed in Experiment 4.3 across the two target conditions, there is an indication that information on the timing of actions relative to external events could be exploited to improve the timing of muscle activity in the affected arm. (This topic is currently being explored by Dr Lee and his co-workers at Edinburgh.)

4) The results of Chapter IV suggest that an emphasis should be placed on setting treatment exercises for the affected arm within the context of bilateral control.

5) Experiment 3.4 indicates that associated reactions caused by a strong sustained contraction of the contralateral flexors can interfere with therapeutic reinstatement of controlled extension of the elbow. (This issue is being investigated at present by Fiona Lough at Loughborough / Cambridge.)

6) The consistent finding of the so-called "unaffected" arm appearing in some way affected, implies caution should be exercised when its capabilities are set as a standard for therapy.

7) At the theoretical level the results of this thesis suggest the hierarchical model of motor control from which treatment regimes are derived, is in need of reassessment. There is no support for Bobath's (1978) suggestion that lower levels of activity are divorced from higher levels, in the stroke patient. In effect the results presented here run contrary to this position.

A general implication of the findings of this thesis for the physiotherapy profession is that by adopting a motor skills perspective, the patient's motor behaviour can be analysed and better understood and treatment methods scrutinised. Future research along these lines should be guided by a maxim best expressed, ironically enough, by Bobath (1978):-

"...there is in every patient some untapped potential for more highly organised activity. The twofold question is how to reach this potential and, if reached, what rational explanation can be given for it. "

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